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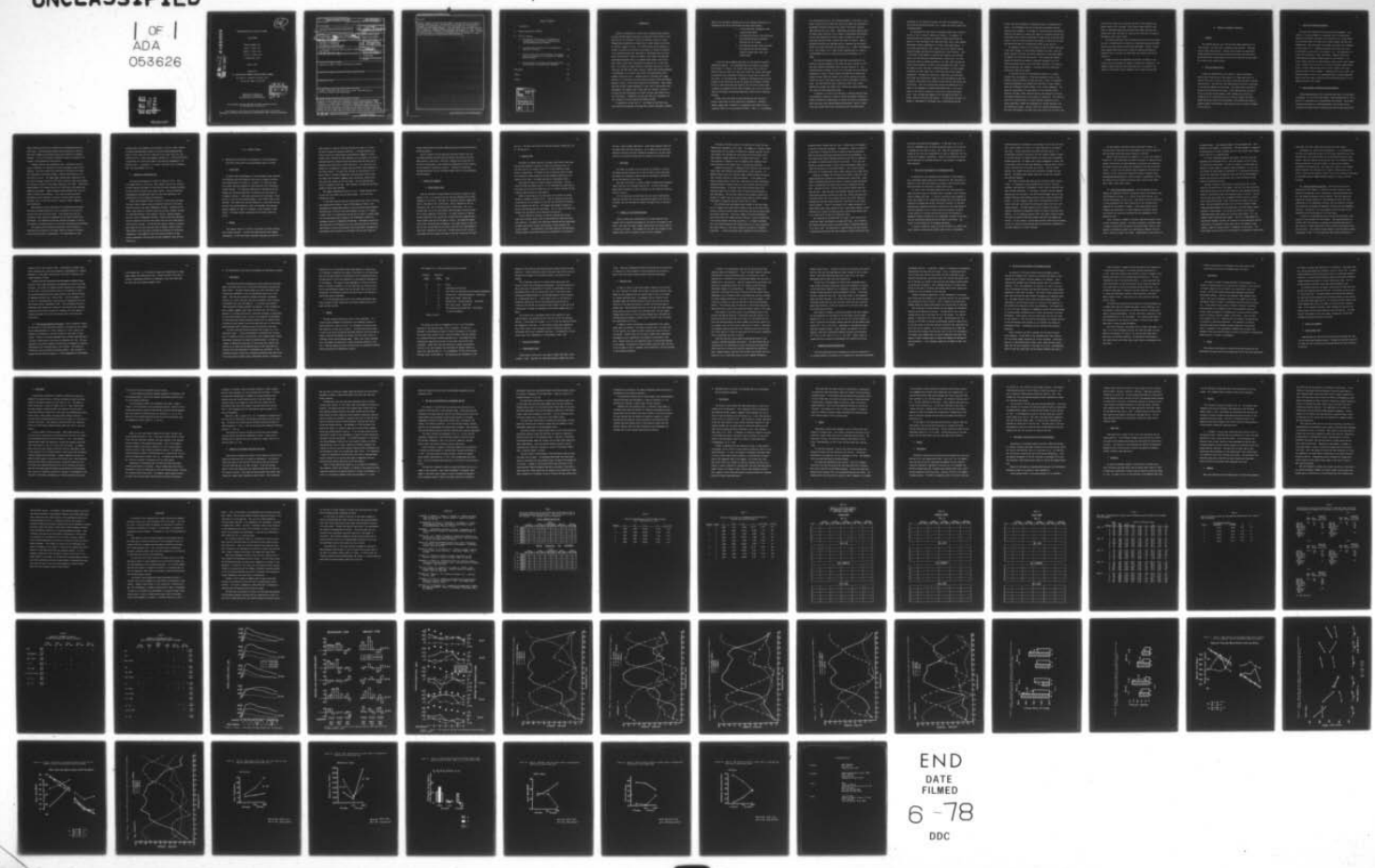

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NEUROPSYCHOLOGICAL STUDIES OF ALCOHOL

FINAL REPORT

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A series of four studies were conducted on highly practiced male volunteer subjects to investigate the relationship of electroencephalographic (EEG) parameters to alcohol-induced performance changes under conditions of low and high motivation and stress. The nature of the EEG-performance association varied with the experimental conditions and was observed primarily when the subject was under high levels of alcohol or was highly stressed or motivated. EEG activation increased when fast performance occurred during a state of high →

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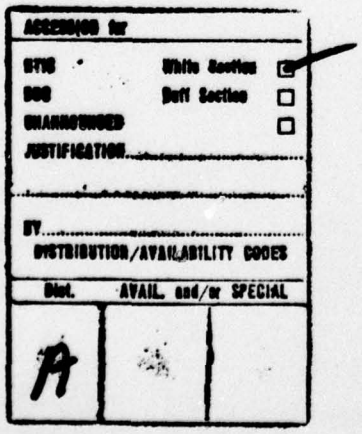
alcohol. Without the state of high alcohol, fast performance was related to decreased EEG activation. The combination of alcohol and sleep deprivation produced interaction effects that varied from antagonistic to synergistic depending on the response measure. From these data it was concluded that the best strategy for looking at EEG-performance relationships is to put a load on the system either in the form of a handicap (high alcohol) or stress. To look for EEG correlates of good and bad performance when the system is in normal condition would seem to require other than scalp EEG.

TABLE OF CONTENTS



I. INTRODUCTION

Alcohol is recognized as a major social problem and much research has been directed toward assessing its acute and chronic effects on the brain and on the individual's ability to perform a variety of tasks. Although much information has been gained, alcohol research has failed to clarify a number of issues. One issue concerns how the effects of alcohol on the brain are related to the effects of alcohol on behavior. It has often been noted that impairment of performance does not consistently follow alcohol intake, for example, some reports indicate that performance impairment occurs at moderate blood alcohol levels while other studies report that individuals are impaired only at very high BALs. Our studies of highly practiced individuals have led us to conclude that the idea that alcohol produces a smooth dose-related decrement in performance is an artifact that results from pooling data across subjects and across trials. Repeated study of practiced, well-known subjects, on a task where one minute blocks provide reliable performance measures, allows a fine grained resolution of performance. These studies show that alcohol related decrements (if any) result from episodic poor performance, and suggests that alcohol does not "produce" a state associated with poor performance but rather increases the probability of poor performance. Furthermore, we have seen that poor performance can occur (though with lower probability) without alcohol.

It appeared to us that this si[illegible]tion might be clarified if we could identify EEG patterns associated with various performance extremes.

Based on the available literature and our own laboratory experience, we hypothesized that three identifiable EEG states might emerge:

1. a non-alcohol poor performance state (possibly high theta)
2. an alcohol-high arousal state associated with high BAL and good performance (possibly low 10 Hz alpha)
3. an alcohol-low arousal state associated with high BAL and poor performance (possibly slowed alpha, high "fast frontal beta").

If such EEG states emerged they might be associated with specific performance patterns. Poor performance EEG state might be associated with "blocks" or "lapses"; the alcohol-high arousal state with fast reaction times but high error rates; the alcohol-low arousal state might be associated with differential slowing of reaction time on more difficult tasks. We considered that if patterns of performance seen with and without alcohol during the various EEG states were different, we might be able to learn more about the specific sorts of errors alcohol tends to produce (as opposed to errors due to fatigue, etc.) and so be able to identify tasks where non-drinking regulations should be most vigorously enforced.

Another issue with which we were concerned was the relation of stress or motivation to brain state and to performance. Anecdotal reports suggest that a traumatic or frightening event tends to have a "sobering" effect on an intoxicated individual. That is, in an emergency

the intoxicated person can "pull himself together" at least for a time. Animal research has also shown that stress can reduce the impairment of psychomotor functions resulting from alcohol intoxication (Leikola, 1962; Wallgren and Tirri, 1963). Experiments with human subjects tend to support these findings, since a number of experimental manipulations such as sleep deprivation, electric shock, extreme cold, and use of incentives have been shown to modify the effect of alcohol. However, the modifying effect has sometimes been to counteract the detrimental effects of alcohol (Wilkinson and Colquhoun, Exp. 1, 1968; Frankenhaeuser, et al, 1974; Korman, et al, 1960) and has sometimes been to intensify the detrimental effect of alcohol (Wilkinson and Colquhoun, Exp. 11, 1968).

Wilkinson and Colquhoun (1968) found that sleep deprivation appeared to sensitize subjects to the effective dose of alcohol so that high doses impaired performance while low doses improved it. They interpreted their findings in terms of an inverted-U function relating performance to level of arousal where sleep deprivation reduced the subjects arousal below the optimal level and the low dose of alcohol raised the arousal level again causing relative improvement in performance. The high effective dose, according to their view, further depressed an already low arousal level causing even poorer performance than seen with sleep deprivation alone.

It appeared to us that it was necessary to measure both EEG state and performance variables in order to clarify the complex interaction effects observed with combinations of alcohol and stress or motivation. Would motivation cause EEG-performance dissociation? Would it simply cause more alcohol-high arousal EEG periods with associated better

performance or can motivation increase the level of performance even during adverse EEG-defined states (e.g., alcohol-low arousal and/or poor performance EEGs)?

We considered that this type of information might have a practical benefit as well as being of theoretical interest. For example, if increased motivation produced its benefits through reducing the frequency of a poor performance high theta EEG state, then a theta feedback device might improve performance in a mildly intoxicated subject. If this effect were observed, such a device also could identify subjects whose performance had been overly compromised by their drinking. Thus, a simple device that measures a specific EEG frequency band could be used to test the effective alcohol-motivation interaction in a mildly intoxicated person to determine whether or not they could overcome the alcohol-induced tendency to enter a poor-performance EEG state. Such a test might be better than BAL in determining whether to allow subjects to proceed with tasks where such a state might have serious adverse consequences. We might also show that a subject who was overtrained while sober could nevertheless reduce his poor performances during mild to moderate intoxication with alcohol by practicing the task during such intoxication. Would any learning that occured be reflected by an increase in the frequency of alcohol-high arousal states, or by an increase in the ability to perform in spite of an alcohol-low arousal state? Thus, we might know whether or not training-with-alcohol is simply training to maintain high arousal. In other words, we could tell whether an improvement of performance due to training-with-alcohol

differs from the improvement of performance caused by heightened motivation. Such knowledge could also be applied to personnel training, selection and assignment. For example, if training-while-intoxicated is effective only because it reduces the number of low arousal periods when intoxicated, then it would be efficient to teach people to maintain high arousal, since this would counteract the effects of alcohol on any task, and obviate the need for training-with-alcohol on each new task.

Our approach in much of this research has been to study intensively a few normal subjects as they perform repeatedly over days, weeks and months instead of the more conventional approach of using large numbers of subjects for relatively few sessions. This approach was adopted because the relations we sought to study were expected to be of an intricate, perhaps subtle, nature and highly individual. We wished to avoid pooling data over subjects, a procedure which we feared would obscure the relations we sought.

In selecting a task for the performance aspect of the studies reported here, we adopted an "information processing" point of view. Recently, human performance has been described in terms of information processing components, and attempts have been made to assess whether there are differential alcohol effects on the various components. The attention requirements of a task appear to be an important factor. Moskowitz and Depry (1968) required subjects simultaneously to monitor and respond differentially to two sources of information. This task was sensitive to moderate doses of alcohol; whereas response to either source separately, without the requirement for divided attention, was not affected by alcohol. Huntley (1973) also required performance on two tasks simultaneously and found that while alcohol did not affect the

high-priority primary task, reaction time (RT) to the secondary task became longer as BAL increased. While these divided attention tasks might be considered simply more difficult than either task when presented alone, task difficulty by itself was not sufficient to produce a detrimental effect under alcohol.

The Huntley experiment also illustrates the notion that the subject has only a limited amount of attention or processing capacity available that must be shared among the tasks being performed. Alcohol or other state changes might produce their effects by reducing the amount of attention that is available or might alter the priority rules for assigning it.

Another variable that determines the effects of alcohol is the speed at which the subject is required to process the information. For example, reduced capacity after alcohol may not be a problem if the subject is given ample time to complete a given stage of processing.

II. GENERAL EXPERIMENTAL PROCEDURES

1. Subjects

The subjects were men ages 22-30 from the student population of a local college. All were light to moderate social drinkers. They were paid for their participation. Subjects were screened for good health, normal eyesight and were asked to abstain from alcohol and other drugs for 24 hours prior to each session, to be well-rested and not have eaten for 4 hours prior to each session.

2. Dose and Administration

Alcohol was administered as 95% ethanol in doses calculated in terms of mg of EtOH per kg of body weight. All doses were given in a base of fruit juice in which a few drops of mint were floated to disguise taste and odor. The particular dose administered varied and is described in the specific methods for each study. The placebo drink consisted of the fruit juice base with mint drops. Drink administration was double blind, as was BAL testing insofar as BAL readings were printed out automatically on a card which was out of sight to subject and experimenter during the course of the experiment. BAL readings were taken by breath by means of the Omicron Intoxilyzer at 6-12 min intervals throughout the session.

3. Tasks and Performance Apparatus

The tasks were presented on the Visual Display Programmer. Five stimulus cells were arranged in a horizontal row on a sloping panel (Industrial Electronics Engineers, Inline Display Cells, Series 10000). Only the three center cells were used in these studies, the other two remained dark. When the subject was seated, the center cells subtended a visual angle of 25°. Responses were made by pressing one of four microswitches on a box held in the subject's lap. The presentation of stimuli was controlled by paper tape reader. Response latency and correctness were recorded on magnetic tape for later analysis. The visual stimuli consisted of simple geometric white outline forms (square, circle, triangle, plus sign and X) on a black background, approximately 20 mm in largest dimension and colored circular patches (red, blue, yellow or green) 27 mm in diameter. Depending on the task, stimuli were presented singly or with a form superimposed upon a colored background. Tasks varied from study to study and are described specifically in the methods section for each study.

4. EEG Electrodes, Recording and Data Reduction

Electroencephalographic (EEG) recordings were made on a Grass Model 6 polygraph. For the first three studies, leads recorded were F3, F4, P3, and P4, all referenced to Cz (international 10-20 system). Analog tape recordings were made on a Crown-Vetter Model A, FM recorder during alternate one minute blocks of performing the task and eyes-closed rest.

These alternate blocks will be referred to as Performance blocks and Rest blocks. Electrooculogram channels were monitored for artifacts, which were flagged on the analog control-pulse channel for automatic deletion. A 10 Hz, 50 microvolt calibration signal was recorded on all channels at the beginning of each session.

Frequency analysis was performed on these recordings using Fast Fourier Transforms in a dedicated real-time environment using a PDP-15 computer. The analog signals were digitized at 128 points per second and transformed in one second windows, yielding coefficients for 1 Hz bins from 0 to 64 Hz. The signals were filtered upon recording at 1 Hz and 70 Hz half-power points and with a 50 Hz low-pass filter upon playback to eliminate "aliasing" of the higher frequencies into lower. Coefficients were averaged for 10 seconds and pairs of coefficients were squared and summed to yield power. Higher frequencies were combined into 2 and 4 Hz bands to simplify data reduction. Thus, each one minute period of Performance or Rest produced 6 successive 10 second power spectral estimates from 1 to 45 Hz for each of 4 channels (higher frequencies were negligible).

These data were further compressed in a program which calculated the mean and standard deviation of the six measures of each spectral variable over each one minute period. This program also used the calibration signal spectra to compensate for any overall channel gain differences. Any remaining artifactual episodes discovered in the on-line graphs of the power spectra or the raw EEG recordings were eliminated.

This program also calculated an additional type of measure to characterize the response of the power spectra to exoteric influences (such as blood alcohol or performance): the second moment of each

spectrum about zero frequency was calculated in each of 5 bands, defined to reflect phenomenological bands of classical electroencephalography: delta (1-3 Hz), theta (4-7 Hz), alpha (8-13 Hz), beta1 (14-21 Hz), and beta2 (22-33 Hz). These second moments, defined as $M = \Sigma f^2 P(f)/(\Sigma f^2)(\Sigma P(f))$ in each band, will reflect the shape of the spectrum independent of its absolute level. In particular, if a peak in the band rises in frequency (Hz), the second moment will rise.

5. Treatment of Performance Data

Each task was presented in a series of blocks of trials. Block size ranged from 20 to 30 trials. Mean reaction time for the initially correct responses and number of errors per block were computed separately for each subject for each block of each session. Various normalizing transformation procedures were used for these data depending on the characteristics of the response distributions.

Except where otherwise noted, analyses of variance were performed with dose, session (when a dose was given more than once), and blocks (within session) as the usual independent variables. Depending on the study, sleep condition, incentive condition and task parameter (divided vs. undivided attention, inter-stimulus interval, response category) were also used as independent variables. Analyses were done separately for each subject for the first two studies with the error term based on within session variance. For the last two studies, subjects' responses were pooled and the error term was based on between subject variance. Simple effects tests were done following the occurrence of significant interactions. Non-parametric tests were used occasionally to test specific hypotheses involving data for which parametric tests were not appropriate.

III. SPECIFIC STUDIES

A. Relationship of EEG State to Performance of a Divided Attention Task during Intoxication by Low and Moderate Doses of Alcohol.

1. Description

We report here on performance of five individuals under conditions of divided and undivided attention, each given on separate occasions three moderate doses of alcohol or a placebo, in a long-term study. Each alcohol dose was presented on three occasions with interspersed placebo sessions. This permitted examination of performance and EEG changes with increasing experience in performing the task under the influence of alcohol. Testing was carried out for three hours in each session, so that we could examine changes in the alcohol effect as BAL declined. The primary task involved response to a tone which occurred on 50% of the divided attention trials; the secondary task required making a judgment of "same" or "different" to two sequential visual stimuli. The delay interval separating the two visual stimuli was varied.

2. Method

Each subject served in a total of 18 sessions (9 alcohol sessions and 9 placebo sessions). Alcohol and placebo sessions were randomly interspersed. Of the nine alcohol sessions, three were at a dose of .9

ml/kg, three at a dose of .65 ml/kg and three at a dose of .4 ml/kg. The task involved two attention conditions: a divided attention condition in which the subject made a same-different judgment to visual stimuli while listening for and responding to an occasional tone, and an undivided attention condition where the subject made the same kind of judgment but was told that no tones would occur. On a tone trial the subject was to respond on a separate key and refrain from responding to the visual stimuli. The other task variable was the duration of interval (248 vs. 632 msec) between the first and second visual stimuli. The "same" and "different" judgments were indicated by pressing keys with one of two fingers on the right hand. The left hand pressed a switch in response to the tone. Equal numbers of divided and undivided attention blocks were presented.

The subject completed his drink in 15 min. Fifteen minutes later he took the first breath test and testing began. Two 10 min rest periods were given.

The EEG data analyzed from each subject were drawn from 18 sessions, each comprising 84 minutes of data (42 Performance, 42 Rest), each represented by average power spectral values and within-minute standard deviations and 5 classical band second moments for each of 4 channels. A number from 1 to 4 representing the quartile of overall response speed was the performance measure chosen as the independent variable for subsequent analysis of dependent EEG measures. Performance ratings for each one-minute of Performance were also associated with the following Rest minute so that any EEG measures found significantly connected with performance could be tested for persistence into the Rest condition.

Values interpolated from the BAL readings were also associated with each one-minute period.

We will present first the data from the blood alcohol level and performance measures and then the data showing the relation of performance and BAL to EEG state. With only 5 subjects and a great deal of variability among them, it was considered inappropriate to pool subjects for the purpose of statistical analysis. Therefore, the data were analyzed separately for each individual subject both for the performance and EEG measures.

3. Results and Comments

a. Blood Alcohol Level

BALs for the mean of three sessons at each dose are shown for each subject in Fig. 1 at successive 10 min intervals beginning 20 min after completion of the drink. Peak BALs for individual sessions ranged from .047 to .083 mg% for the .9 ml/kg dose, .035 to .060 mg% for the .65 ml/kg dose, and .010 to .029 mg% for the .4 ml/kg dose. Fig. 1 indicates that, for all but one subject, BALs had begun to decline within 40 min after completion of the drink. For these subjects all testing periods except the first one for the .9 ml/kg dose were conducted while blood alcohol level was at the peak or falling. The remaining subject had a slowly increasing BAL followed by a long stable period so that his BAL for the .9 ml/kg dose did not begin to decline until approximately 2 hours after completion of the drink. The BAL curves for the .65 and .4 ml/kg doses for this subject also show a slow rate of decline (.007 mg%

per hr.). The rates for decline for the other subjects ranged from .014 to .017 mg% per hr.

b. Reaction Time

The effect of highest dose was to produce significantly lower mean RTs than the placebo on from one to three of the sessions (Fig. 2). This was true for all subjects except for S#3 who showed no alcohol effect at these doses. The effect of the low dose was either to speed RT or to have no effect except for S#3 who produced slower RTs on one session at this dose. The middle dose had an intermediate effect.

Over days the effect of alcohol gradually diminished. This was true for either the highest dose or, if the impairment was maintained at that dose, was true for the middle dose for all subjects (except S#3 who failed to show an effect at these doses).

Within a given session, the time course of the effect of alcohol varied for different subjects (Fig 3). Peak RT slowing occurred early in the session for two subjects (#1 and #5) and occurred at mid-session for two subjects (#2 and #4). The highest dose tended to produce an earlier peak slowing than the lower doses. For all doses, the effect of alcohol on RT was nearly zero by the end of the session.

It was expected on the basis of previous research that alcohol would have a greater effect on RT in the divided than undivided attention condition. In general this effect was observed but was complicated by being restricted to certain test periods or delay intervals for various subjects. The combination of divided attention and short delay interval appear to have been particularly susceptible to the impact of

the drug. Under placebo conditions the short delay produced faster RTs and fewer errors than the long delay. So it seems that the conditions leading to the most efficient information processing in the non-alcohol state are not necessarily those that are optimal in the alcohol state.

c. Error Rate

The effect of alcohol on error rate was to increase it in one of the three high dose sessions but to show no effect at the lower doses. Facilitation of accuracy by the low dose was considered a possibility but was not observed in this study.

Over days, the effects of alcohol tended to become more detrimental rather than less so as had been found for RT. As with RT the short delay interval tended to be more susceptible to the impairing effects of alcohol on errors.

It is of particular interest that the only sign of deterioration under the high dose for S#3, who consistently showed RT facilitation or no effect, was the occurrence of greatly increased errors at the short delay.

d. Comments on the Performance Data

Several studies have indicated that on divided attention tasks subjects tend to maintain performance on one task at the expense of the other. One might also expect unequal deterioration in the two tasks as a function of alcohol. This tendency was not seen very strongly in the present study since it occurred in only 2 of the 5 subjects.

The highly individual nature of the reaction to alcohol has been demonstrated repeatedly in this study. For example, all subjects showed some tolerance to the effect of alcohol over repeated sessions; but for some subjects the tolerance was restricted to the lower doses, while other subjects showed tolerance at the highest dose as well. An obvious question is whether or not all subjects would eventually have shown tolerance to the highest dose if sessions had continued.

Subject #3 was peculiar in showing RT facilitation at the high and middle doses while showing some deterioration at the low dose. His behavior might be due to an internal state condition related to arousal that interacts with BAL to affect performance. If S#3 tended to be highly aroused without alcohol then a high dose of alcohol would have a depressing effect, bringing arousal to a more optimal level and facilitating performance. A low dose, which often has been found to be arousing, might push the arousal level for S#3 too high resulting in poor performance. The idea that highly aroused subjects respond differently to alcohol than less highly aroused subjects receives support from reports reviewed by Wallgren & Barry 1970 (p. 356).

The relatively poor performance under alcohol at the short delay for all subjects was of particular interest insofar as the short delay conditon resulted in better speed and accuracy than the long delay under non-alcohol conditions. There are a number of possible explanations. At the short delay the subject had only 248 msec in which to encode the first stimulus before the second one came on. If alcohol interfered with this encoding operation the subject would have to delay processing the second stimulus or else would respond on the basis of incomplete processing. To delay processing would lengthen the RT while partial

processing would increase the error rate. Subjects may have engaged in a mixture of these two strategies. Alternatively, lapses of attention or difficulty in timing the shifts in attention from one processing phase to the next would delay processing and disrupt performance. At the longer delay interval encoding would have more time for completion and lapses in attention would also be less serious. To be sure, the slowing of encoding posited in some of these explanations ought also to affect the processing of the second stimulus. But a double slowing would occur at the short delay and a single slowing at the longer delay.

There was a tendency for the effects of alcohol to decline as the subject gained more experience in performing while intoxicated. Jones (1972) has demonstrated that the effect of alcohol on cognitive performance is reduced when the subject has had prior experience with the task, provided the task is one for which practice effects would normally be seen. Since the task in the present study was one for which large practice effects occurred and since the subjects had many hours of practice before the first alcohol session, the alcohol effects we observed may have been reduced relative to the effect on less highly trained subjects. Three of the subjects still showed substantial alcohol effects after three sessions at the .9 ml/kg dose, however, so the interaction of alcohol with practice may be dose-related and may level off at some fairly low but still above-zero level.

One conclusion to be drawn from this study is that low doses of alcohol may impair performance in unpredictable ways. We can observe but cannot accurately predict occasional lapses of competence when BAL is as low as .05%. The implication is that we should be very cautious in permitting persons who have been drinking to perform tasks which have

high costs associated with incompetence. On the other hand, if the costs of incompetence are not great, we may get quite a lot of work out of someone who has a BAL as high as .10%. Much more investigation is needed not only into the susceptibility of tasks to impairment, but into the nature of temporal fluctuations. Study of the possibility of overcoming impairment by increased motivation is one approach to answering these questions.

e. EEG and its Relationship to Performance and BAL

As discussed in the introduction the intention of these analyses was to determine whether there exist characteristic EEG "signatures" or patterns of tonic CNS states which are related to good and poor performance and detect EEG indicators of blood alcohol level.

In order to make a thorough exploration of the relationships between the three classes of variables (EEG, RT and BAL), three different analytic methods were used: (1) partial correlations were computed to assess the strength of the relationship between EEG and RT (holding BAL constant) and between EEG and BAL (holding RT constant); (2) Multiple regression was computed to provide an indication of the total amount of variance accounted for by all three classes of variables; (3) Multivariate analysis of variance was carried out with EEG variables as dependent variables and BAL and RT as independent variables in the event that the relationship between EEG and RT might be non-linear. The results of each method will be presented separately.

In order to reduce the large set of EEG variables to a smaller set, factor analysis was done using spectral power values, corresponding

standard deviations and moments for each channel on half the total data from a given subject at a time (i.e., every other pair of one-minute data sets). Several conclusions were drawn from the results: (1) corresponding variables from each of the four channels tended to appear on the same factors; (2) powers tended to be paired with corresponding standard deviations; (3) moments were largely independent of powers and of each other, those for each band commanding a separate factor; and (4) factors from each half of the data were similar. Thus for further analyses, the standard deviations were considered redundant and were omitted. The moments were treated separately, and the four channels were analyzed together.

Next factor analyses were done two ways using only power spectral values: (1) separately on each combination of lead pair (frontals, parietals) and condition ("Performance", "Rest"), and (2) over both lead pairs and both conditions. If the factor structures from the separate analyses were too dissimilar, then the variance between lead pairs and between conditions would dominate overall factors, masking interesting effects. For four subjects, these factor structures were similar, so factor scores were calculated from the overall factors for use in subsequent analyses. The power spectral factors tended to be bands of adjacent frequencies corresponding roughly to classical EEG bands (see Results). For the remaining subject (S#2), the overall factors largely reflected the extreme differences between lead pairs and between conditions. For this subject, average power spectral values were calculated in the five classical bands defined above to provide data comparable to the other subjects for further analyses.

For each subject, the factor scores from overall factors (or classical bands values) and the second moments from classical bands for each one-minute period were used as measures of the EEG.

Overall factor structures for subjects 1, 3, 4 and 5 are plotted in Figures 4-7. (As noted above, the factor structures for subject #2 were complex and dissimilar in each combination of lead pair and condition; the overall factors were simple and represented little but the dissimilarity.) In most cases the spectral power frequencies achieving high loading (>.5) on a given factor comprise contiguous frequency bands corresponding approximately to classical bands or combinations of them. The factors will be referred to below by those classical band names: delta, theta, alpha, beta1, beta2.

(1) Partial Correlation Analyses - Each EEG measure for each subject was subjected to partial correlation analysis with blood alcohol and performance variables, i.e., partial correlation with BAL, controlling performance, and vice versa. The method of partial correlation is used to determine the linear association of two variables while adjusting for the effects of another. Since blood alcohol and performance are (potentially, at least) themselves correlated, partial correlations are required to determine the true dependence of EEG measures on each.

Table 1 presents a summary of the most significant results of the partial correlation analysis. Each entry represents a variable, factor or moment, whose partial correlation with blood alcohol level or with quality of performance proved to be significantly different from zero with $p < .001$ in at least 2 of 4 leads. Interpretation of the factors is

straight-forward -- they represent power in the designated band. Interpretation of the moments is more complex. The second moment is responsive to the position of a peak if present, but will also reflect other changes such as the tilt of a peakless band.

It is most immediately apparent from Table 1 that few of the EEG parameters are correlated with RT. Only two subjects (#1 and 2) show any significant correlations. For S#1, an increase in alpha moment and decrease in betal moment is related to good performance. For S#2, the relationship of alpha moment to performance is in the opposite direction. For S#1 the significant correlations occur only during the performance minutes, not during the rest periods, and so must be related to some aspect of performing the task.

The high incidence of significant correlations with BAL is indication of the sensitivity of the method. Note too that the correlations found tend to persist into the "Rest" condition; they represent organismic states of intoxication rather than transient states of arousal. Despite the fact that each subject has a somewhat different pattern of significant correlations with BAL, a few correlations are common to most subjects. During performance minutes, four of the five subjects showed increasing theta moments with increasing BAL, and three subjects showed reduction in betal moment. During the rest minutes, four subjects showed decreasing alpha moment and increasing theta moment. All five subjects showed decreasing betal moment. The rise of theta moment with elevated BAL probably signifies the invasion of the conventionally defined theta band by the generally higher power peak of the subjects' alpha rhythms, slowed (or perhaps spread in frequency) by the alcohol. The betal moment results accord with the hypothesis of broadened or destabilized

alpha peak; the "Rest" alpha moment reflects net alpha slowing.

Correlations for factors were idiosyncratic. It is interesting to note that even though the variance for RT has been "partialled out", the relationship of EEG to BAL differs for the performing minutes and rest minutes. When not required to perform four out of five subjects showed EEGs characteristic of the early stages of sleep and this tendency increased with increasing BAL. When required to perform, this tendency toward sleep was less strong but still present. Another point of interest is that S#3 (who tended to show RT facilitation instead of impairment at the highest doses), showed decreasing amounts of alpha as his BAL increased.

(2) Multiple Regression Analyses - Multiple regressions were performed (for each subject) to ascertain the total extent of linear association between BAL and all EEG measures, between performance and BAL, and between performance and EEG measures. Multiple regressions provide the optimum prediction of the dependent variable from linear combinations of the independent variables, and thus determine the multiple correlation (R) between the dependent variables and the independent variables. The square of R is the proportion of the variance of the dependent variable accountable by the linear model.

In Table 2 are presented the results of multiple regression of BAL against all EEG variables for each subject. The squared multiple correlation (R^2) thus obtained is a measure of the maximum proportion of the variance of linear combinations of EEG variables predictable by BAL, and vice versa. Thus alcohol produced changes in the EEG pattern roughly proportional to BAL -- to the extent that EEG variables predicted an

average of 50% of the variance of BAL. The patterns of change, while quite individualized, could be most generally comprehended as a reduced abundance of alpha power, particularly in the "Rest" condition, and a slowed frequency of alpha.

Stepwise multiple regressions were performed to determine those portions of the linear dependence of performance due solely to the EEG variables. The results of stepwise multiple regressions of performance vs. BAL (step 1) and EEG variables (step 2) are displayed in Table 3. The multiple regression process takes account of the intercorrelations of dependent variables (e.g., BAL and EEG). Thus the increment in R^2 in step 2 is that proportion of the variance of performance due to the EEG variables alone, independent of BAL. Thus performance was poorly predicted by BAL (average of 4 percent of the variance) and, although adding the EEG variables improved the situation, the final amount of variance accounted for was only 15 percent and of little practical significance.

(3) Multivariate Analysis of Variance - Table 4 presents a summary of the results of the multivariate analysis of variance for each subject which tested hypotheses of linear, quadratic, or cubic trends of dependence of performance on the EEG variables at each level of blood alcohol. Entries in the table represent those variables contributing to an overall significance of the particular hypothesis of .001. The sign appended to each entry indicates the direction of dependence of improved performance on the indicated power of the variable. The scatter of significant results manifests no coherent pattern. Only one entry is present for more than two subjects: a linear dependence on theta moment

at the highest BAL. It is difficult to make any interpretation of these highly complex and idiosyncratic data. Perhaps the brain state that is related to performance variables is organized at some level other than the level that that produces changes in EEG.

B. The Relationship of EEG State to Performance at High Doses of Alcohol

1. Description

The preceding study had produced only slight indication that knowledge of EEG state would assist in predicting performance deterioration under alcohol. The major problem seemed to be that impairment of performance was problematic at the low and moderate doses used in that study. There was much variability between individuals and between sessions for a given individual that may have masked a subtle relationship between brain state and behavior. To alleviate that problem a "task-controlled drinking procedure" was adopted. In this procedure we gave a subject repeated, small doses of alcohol until the first appearance of a minimum statistically significant decrement in performance on the divided attention task, using an on-line measure of performance. The task-controlled drinking procedure assured that all subjects would be tested at blood alcohol levels which were sufficient to cause performance decrements while avoiding the use of arbitrarily large doses.

The task-controlled drinking procedure also allowed us to study additional issues. 1) Once the decrement in performance was achieved, would it continue as long as blood alcohol levels remained high or would performance fluctuate as we found with moderate doses? 2) Would the tendency to demonstrate impairment at these higher BALs diminish over sessions as we found with the moderate doses or were these BALs sufficiently high that impairment of performance was inevitable? 3) The attention variable of the visual information processing task used in the first study did not produce clearly differential effects in response to

alcohol but such a differential effect might emerge at a higher dose. 4) In addition to obtaining the subject's evaluation of his intoxication level we also would obtain his prediction as to his performance during the next block of trials and afterwards his retrospective assessment of his performance. The study of these assessments in relation to performance is relevant to whether or not the individual can correctly evaluate his own performance capabilities and may shed light on short term fluctuations in performance capacity that occur even in the absence of significant overall impairment.

As in the preceding study we will first present performance data alone and then the data showing the relationship between EEG, performance variables and BAL.

2. Method

The same subjects continued to serve in this experiment. The visual display programmer was modified to give immediate read-out of reaction time for a block of trials. The information processing tasks were identical to those used in Study I. Following two warm-up blocks on each of the tasks (divided and undivided attention) which provided an index of baseline performance for the day, the first 15-min cycle of drinking, ratings and performance began. These 15-min cycles continued until the subject had received 6-7 drinks (containing placebo or alcohol.) Then the subject was given no more drinks but continued to perform and give ratings for an additional 8 cycles.

The schedule for a 15-min drinking cycle was as follows:

Duration (min)	Cumulative Time	Task
3-5	3-5	Drink.
4	9	Rinse mouth out and rest.
1	10	Self-rating of intoxication and predicted performance.
1	11	Perform task - Undivided attention. Record EEG.
1	12	Rest, eyes closed. Record EEG.
1	13	Perform task - Divided attention. Record EEG.
1	14	Rest, eyes closed. Record EEG.
1	15	BAL recording from Intoxilizer. Self-rating of past performance.

Return to step 1.

The alcohol was given in increments of 1/6 or 1/3 of the highest dose used in the previous study (.07 to .24 ml/kg). As soon as a decrement in performance was noted in the on-line measure, or else when the subject's BAL reached .10 to .12 g/% the alcohol drink was replaced by a placebo drink until 6 drinks had been given. An exception to this procedure was required for S#1 who, at this time, took on full-time employment. Rather than lose him entirely as a subject we decided to test him in the early evening, but restricted his alcohol intake to a lower level than the other Ss. As a consequence, his BALs did not reach the high levels of the other Ss. The subject was not informed as to the

contents of the drink but knew that some would contain alcohol and some would not. The BAL reading was taken at the end of each 15-min cycle to minimize the influence of any residual alcohol in the mouth on the reading.

The 1/3 dose was used for all alcohol drinks during the first session at which time a problem was encountered. With some subjects, at least, there was a delayed effect of alcohol on performance so that a performance deficit did not show up until later in the session after drinking had stopped (because of the maximum number of drinks required for a performance deficit). To gain better control of the period of increasing intoxication and to allow performance deterioration to "catch-up" with the BAL, we altered the dose schedule so that the first 3 drinks were 1/3 doses and subsequent drinks (where needed) were 1/6 doses.

The criterion for a performance deficit was computed for each session based on the baseline data for that day and for the preceding session. A value based on the mean +1.5 S.D. of the two baseline sessions was computed for each task. If the criterion values were exceeded on two of four blocks in two successive cycles of performing, then the performance deficit was considered significant. The probability of such values under a null hypothesis of no impairment is about .008.

3. Results and Comments

a. Blood Alcohol Level

Blood alcohol levels did in fact reach a higher level than in the preceding study. Peak BALs for individual sessions ranged from .06 to

.12 g%. There was considerable variability from session to session due in large part to the fluctuation in dose reflecting the variation in dose at which the subject showed minimum significant impairment.

b. Reaction Time

As shown in Table 5, significant overall slowing of RT was found for 5 of 6 sessions for either the divided or undivided attention tasks, except for S#1 (whose BAL did not reach as high a level as the others for reasons explained above). In agreement with our results in the preceding study, the divided attention task did not prove to be more sensitive to the impairing effects of alcohol than the undivided attention task. Mean performance deficits for the two tasks were compared across sessions using t-tests (for correlated means) for each subject. Although the direction of the difference between tasks indicated that divided attention produced a greater deficit (Table 6) this difference was significant only for S#1.

A number of factors contributed to the magnitude of the alcohol effect which was observed. One group of factors were: the total dose for the session, the schedule by which the dose was given and the resulting BAL. Another factor was the session number which represented the amount of prior experience of the subject in performing at that dose. Another factor was the subjective level of intoxication reported by the subject. Correlations of these variables with each other and with RT as are shown in Table 7 (Spearman rank correlations), and are discussed in the following paragraph.

In Table 7, the performance scores for the two tasks have been pooled to make the "composite RT". Due to the small number of sessions contributing to these correlations (N=6), a correlation of .829 is required for a correlation to be significantly greater than zero with a probability of .05 (one-tail). BAL had a high positive correlation with magnitude of performance deficit for all subjects except S#2 whose performance tended to be unpredictable. Performance tended to be negatively correlated with session indicating that the alcohol effect (magnitude of the difference scores) diminished over sessions, although the trend reached significance in only one of the five subjects. Self-rating was no better or was worse than BAL at predicting performance deficit. The relatively poor correlations at self-rating with performance may be due in part to the restricted range of doses in this study.

With respect to the time course of the performance deficit, the earliest test period producing a significant effect was period 5 or 6 (occurring 75-90 min after the first drink); for the divided task the first test period producing an effect tended to be earlier, period 3 or 4 in several cases (at 45 to 60 min after the first drink.) There were also numerous sessions where the first significant point did not occur until after period 8 (2 hours or more after the first drink), especially with the undivided task for Ss 4 and 5.

There was only one occasion when the performance deficit, once achieved, continued throughout the session. The usual pattern was for isolated points of significance to occur with several non-significant (often near zero or below zero) deficits separating the significant ones. Another pattern, seen most often on the first session, was for a cluster of 2 or 3 significant points to occur together followed by

gradual return to zero. The BALs at the end of the session were usually near the .06% level and sometimes well above (except for S#4 as noted above). Even when terminating BALs were at the .08 to .10% level, performance deficits were seen only sporadically.

The ability of the subjects to predict their performance while under alcohol and under placebo was assessed by performing chi squares on the predictions (combined into 3 categories: prediction of above average, average and below average performance) and RTs. For placebo sessions, average RTs were used. These were placed in two categories (above and below the mean). For alcohol sessions, the alcohol-placebo difference scores were used and these were classified into a positive group (above 26 msec), a near zero group (-25 to +25 msec) and a negative group (below -26 msec).

Only subject #1 showed a significantly greater than zero tendency for his predictions to correspond to his performance during placebo sessions ($\chi^2 = 4.7$, $p < .05$, df=1) and S#1 was also the only subject to show a significant relationship between prediction and performance under alcohol ($\chi^2 = 9.5$, $p < .05$, df=4). Assessment of retrospective performance was treated similarly. Under placebo, two subjects (#1 and 4) showed a significant relationship between assessment and performance ($\chi^2 = 10.3$, $p < .01$, df=2; $\chi^2 = 5.1$; $p < .05$, df=1). Under alcohol no subject was able to assess his performance with significant accuracy.

c. Comments on the Performance Data

The task-controlled drinking procedure was relatively successful in its primary purpose of providing a high probability of obtaining significant

performance deficits. In addition, a number of interesting and unexpected observations also arose from this study. First, a significant deficit did not occur until the 7th test period (105 min of testing) on more than half the sessions, and did not occur until the 9th test period (135 min of testing) on a fourth of the occasions even though drinking ceased on the 6th and 7th periods. Thus, although BAL was at its peak within 85 min of the start of drinking, performance deterioration lagged after peak BAL by 20 to 50 minutes on many sessions.

Another interesting observation is that maintenance of a high BAL, while increasing the probability of significant deficit, did not guarantee the continuance of such a deficit. This is shown by the fluctuations between normal and impaired performance that occurred even at high BALs.

One of the most striking findings was the tendency for performance deficits to be reduced over sessions. The mean deficit for a session reached zero on one or both tasks for 4 of the subjects. For the 5th subject, it declined to less than 10 msec. The explanation for this decline in the effect of alcohol on performance cannot be insensitivity of the task since, on the first session, the deficit was significant for both tasks for all subjects. Nor can the explanation be a higher BAL during the first session since that was true for only 2 of the subjects. The subjects must somehow learn to perform competently when under high doses of alcohol although there is always the tendency for episodes of poor performance -- 4 of 5 subjects showed such episodes on their last session.

d. EEG and its Relationship to Performance and BAL

The analysis of EEG data differed from the preceding study by pooling the 5 subjects and 6 sessions to ascertain the effects of performance on EEG variables that were common to all subjects. Factor analyses were performed upon the power spectral data of all subjects combined. First, each combination of lead pair (F's and P's) and condition (Performance and Rest) were analyzed separately to assure general congruence of the factor structures. Then overall factor analyses were done, thus providing the same variables throughout the subsequent analysis. The power spectral factor scores and second moments were then subjected to a multivariate analysis of variance to test hypotheses of main effects and interactions of BAL and performance. EEG channel (F3, F4, P3, or P4) was included as an independent variable to test hypotheses of frontal-parietal differences and of frontal and parietal left-right differences. Blood alcohol level was partitioned into only two levels for this analysis. Only two levels of performance, the extreme quartiles of the distribution, were selected so as to maximize the chances of performance effects. Performance and Rest conditions were analyzed separately.

Figure 8 presents the factor loadings from the factor analysis across subjects. As was found in the preceding study, high loadings fall into bands roughly equivalent to clinical EEG bands. Accordingly they will be designated below as delta, theta, alpha and beta factors. (Note that low beta frequencies, from about 16 to 22 Hz, load moderately highly on both the alpha factor and the higher frequency beta factor.)

Table 8 presents a summary of those main effects and interactions in the multivariate analysis of variance reaching significance at $p < .001$. Each entry reflects those variables, factors or moments, which reached significance in the corresponding univariate test. The sign indicated the sign of the univariate effect. It can be seen that the significant effects of high blood alcohol are to lower beta power and raise theta power in both the Performance and Rest conditions. Additionally alpha power is reduced in the Rest condition. Theta moment is raised and betal moment lowered in both conditions, with alpha moment lowered in Rest. These results are fully consistent with the previous study.

The significant EEG patterns associated with rapid performance are increased theta power and reduced beta power in both conditions, with the decrease in beta persisting into the Rest intervals following episodes of improved performance. The only significant interaction of BAL and performance was in the alpha moment. This indicates that there is more slowing due to the presence of alcohol in states of poor performance than in states of good performance.

Left-right differences are present only as higher theta power in P4 than in P3 in the Performance condition and lower beta power and beta moment in Performance in F4 than in F3, with the beta power effect persisting into the Rest condition. No significant interactions of left-right effects with either blood alcohol level or performance level were found.

C. Effects of Motivation on Performance, Tonic Heart Rate and EEG State during Intoxication at Moderate Doses of Alcohol.

1. Description

The effect of alcohol on human behavioral and physiological responses is determined not only by dose and the individual's prior history of alcohol usage but also by factors such as motivation, stress and anxiety. Few alcohol-related studies have attempted to manipulate motivational effects. Wilkinson and Colquhoun (1968) employed incentives in the form of knowledge of results in an attempt to alter the detrimental effect of alcohol on performance and were partially successful. In the present study we used a financial incentive to motivate good performance in an attempt to see if the detrimental effects of alcohol could be reduced or eliminated and if such motivational effects would be related to specific changes in EEG pattern. Heart rate was also recorded since it is responsive both to alcoholic intoxication and to stressful or motivating conditions. This enabled us to look for covariation among three different systems (behavioral, CNS and autonomic) in response to the experimental treatments and to explore the hypothesis that physiological arousal reactions are involved in counteracting adverse effects of alcohol on performance.

2. Method

Nine subjects participated in several pre-testing sessions and four experimental sessions, each session consisting of one of the four combinations

of alcohol or placebo and incentives or no incentives. The ethanol dose was .8 ml/kg body weight and resulted in a BAL of .06 to .08%. A complex divided attention condition was used as well as an undivided attention condition. For the undivided attention condition the subject performed a same-different task alone or a visual search task alone. For the divided attention condition, both tasks were performed at once.

On incentive sessions the subject received 25¢ for each block that met both the speed and accuracy criteria. The accuracy criterion was 0 or 1 error per block. The speed criteria were established separately for each task for each subject during training sessions so that from 20 to 30% of the blocks would pay off. On incentive sessions subjects were given feedback each block as to number of errors and total speed per block. On non-incentive sessions, verbal feedback was not given and subjects were told that there would be no pay-off that day. The total session lasted about 2 hours with breaks interspersed to permit BAL readings and subjective ratings to be made.

3. Results and Comments

a. Blood Alcohol Level

Mean peak BAL was .069 for the alcohol/incentive session and .068 for the alcohol/non-incentive session. The peak was reached at about 60 min from the start of drinking and occurred during the first two blocks of testing.

b. Performance

A significant interaction of alcohol by incentive by blocks was obtained for reaction time ($p < .05$) and a marginally significant interaction of the same variables for error rate ($p < .10$). The nature of these interactions is shown in Figures 9 and 10. These figures show that when subjects received placebo, the incentive condition had no significant effect on either RT or error rate and performance was stable across blocks. When subjects received alcohol without incentives, reaction time was impaired during the first five blocks ($p < .05$) but not the last five blocks. When subjects received alcohol with incentives, RT was not significantly impaired during either the first or last five blocks.

Accuracy showed a different pattern. When alcohol without incentives was given, accuracy was not impaired during the first five blocks but was impaired during the last five blocks ($p < .01$). When subjects received both alcohol and incentives, accuracy was impaired during the first five blocks ($p < .05$) but not the last five blocks. In summary, when alcohol was given without incentives there was impairment of both RT and accuracy, but at different points in the session with an apparent trade-off between the two measures. Subjects appeared to be able to maintain either speed or accuracy but not both at once. When incentives were given with alcohol, again there was a trade-off during the first five blocks with speed maintained at the cost of accuracy, but during the last five blocks good performance was maintained for both measures. Thus, the use of incentives resulted in a net gain in performance in the last half of the session although was of limited assistance during the

first half of the session when BAL was at its peak.

The two attention conditions produced substantial differences in RT and accuracy overall, but did not interact significantly with the alcohol and incentive conditions.

The relationship of BAL to RT performance over time is shown in Figure 11 for the incentive and non-incentive sessions. Mean RT for the alcohol/non-incentive session follows the BAL curve and the two measures were significantly correlated across the 10 blocks ($r = .76$, $p < .01$). Mean RT for the alcohol/incentive session did not follow the BAL curve and appeared to be more stable ($r = -.58$, ns).

c. Heart Rate

Heart rate was significantly higher during alcohol sessions than during placebo sessions ($p < .05$). There was no overall effect of incentives, but when individual subjects' data were examined it was apparent that two patterns of HR response were present. Half of the subjects (group A) showed a significantly greater HR on the alcohol/incentive session relative to the alcohol/non-incentive session. The remaining subjects (group B) showed no HR difference due to incentive but only the usual HR increase due to alcohol (Fig. 12). On placebo sessions there was little or no effect of incentive on HR for either group.

Examination of performance data indicated that these two groups differed significantly in accuracy. Group A showed fewer total errors than group B across all conditions ($p < .05$) and reached the error criterion for incentive pay-off more often. The tendency toward greater accuracy by group A was also consistent during pre-test sessions and probably

represents a relatively stable performance tendency of these subjects. Reaction time performance for the two groups did not differ. It should be noted that although group A appeared to be physiologically more aroused during the alcohol/incentive session than the alcohol non-incentive session (as indexed by faster HR), this arousal did not consistently facilitate their speed or accuracy during that session. That is, HR fluctuations did not consistently relate to speed or accuracy fluctuations.

Mean HR vs. BAL is shown in Fig. 13. HR gradually increased during the first hour of testing and then gradually declined during the second hour. HR and BAL were significantly correlated during the alcohol/incentive session ($r = .69$, $p < .05$) but not during the alcohol/non-incentive session ($r = .43$, ns).

Self-ratings of tension, fatigue and intoxication were obtained from the subjects during each experimental session. Groups A and B differed only on tension level with A reporting a higher level for all sessions than B ($p < .05$).

d. Comments on Performance and Heart Rate Data

These results indicate that, within limits, monetary incentives can reduce the impairing effect of alcohol on performance. The incentive effect took place on the last half of the session but not during the first half when BAL was at or near its peak. It was the accuracy measure rather than speed which was impaired during the first hour of the incentive session. Both speed and accuracy were quite stable under placebo but showed large fluctuations under alcohol. The fluctuations

took the form of trade-offs between speed and accuracy with one measure performed at normal or better-than-normal level while the other was greatly impaired.

Although heart rate has often been reported to show a reliable increase under alcohol, in this study, heart rate showed two patterns of response. One pattern was heart rate increase under alcohol only for the incentive condition (group A) the other pattern was HR increase under alcohol for both incentive and non-incentive conditions (group B). These two groups also differed in accuracy level over all sessions with group A being more accurate and earning more incentives than group B during the placebo sessions. One hypothesis is that the heart rate pattern for group A is related to some kind of extra effort expended during the alcohol/incentive session. If so, the extra effort was not consistently related to any improvement in speed or accuracy during the time heart rate was accelerated. An alternate hypothesis is that heart rate and performance in group A are not causally related, but rather, some personality trait or other individual difference parameter contributes to both the consistent tendency toward accurate performance and the differential heart rate response when under stress. This hypothesis is partially supported by the observation that group A reported higher tension ratings for all sessions than group B suggesting that they experience more tension when under stress than group B.

None of these observations appear to us to support the hypothesis that autonomic arousal (HR increase) is involved in the process by which incentives counteract the adverse effects of alcohol on performance. If there is a relationship between arousal and performance it is more

subtle and complex than the rather straightfoward hypothesis that we selected to test.

e. EEG and its Relationship to Performance and BAL

The analysis of the EEG data was carried out by pooling across subjects as had been done in the preceding study. Factor analyses were performed on the power spectral data and the resulting factor scores and second moments were subjected to multivariate analysis of variance. Independent variables included blood alcohol level, performance, EEG channel, and incentive condition. As in the previous studies, extreme quartiles of the performance distribution were selected. EEGs collected from the Performance condition and Rest condition were analyzed separately.

The factor loadings are presented in Fig. 14. In this case the contiguous frequencies of theta and alpha combine to make one factor. The low beta frequencies, from 14 Hz to 26 Hz, comprise a separate factor, beta1, while delta and beta2 factors again occur.

Table 9 presents a summary of those main effects and interactions in the multivariate analysis of variance which reached significance at $p < .001$. Each entry comprises those variables, factors or moments, which reached significance of $p < .001$ in the corresponding univariate test. The sign associated with each entry is the sign of the univariate effect.

The addition of monetary incentive caused significant effects on a number of EEG variables: beta1 power and moment is increased, delta moment is decreased (which probably reflects increased eye movement, with consequent change of shape of the power spectrum in the delta),

theta moment lower and alpha moment higher (which both probably reflect an increase in frequency of the alpha peak). These are signs of increased activation in the CNS.

The significant interaction of incentive with blood alcohol level in betal power and theta and alpha moments show that more incentive-induced arousal occurs for high blood alcohol levels than for low ones. The theta-alpha power factor was decreased by incentive at high blood alcohol levels but increased at low levels. This too indicates relatively more arousal due to incentive at high alcohol levels.

The significant interaction of incentive and performance on delta moment may indicate that incentive induces more eye movement in high performance states than in low performance states.

The overall effect of blood alcohol level is again associated with significant effects on many EEG variables; the pattern is generally consistent with that of the preceding study -- high BAL is associated with decreased betal power and slowing, and by higher theta moment and lower alpha moments. Again the effects generally persist into the associated Rest condition, indicating a state of intoxication rather than a transient change in arousal.

The main effects of performance in the Performance condition were once again few in number. The decreased betal power with high performance is consistent with the preceding study; decreased theta moment was also found. A number of significant effects were discovered in the associated Rest condition samples that were not present in the Performance condition. These effects may be related to the processing of feedback information as to whether or not an incentive had been earned (this

information was provided to the subject immediately after each block of performance on the incentive sessions).

The significant interactions of blood alcohol level and performance indicate that when high performance is found at high BALs it is associated with increases in Betal power and theta slowing.

Compared with the preceding study, a relatively large number of variables show significant effects of incentive, performance and their interactions with one another and other variables in the Rest condition. This may indicate that the addition of monetary incentive in certain sessions and the generally higher performance correlated with that incentive may have induced states of more general arousal than the previous studies, which are then reflected by their detectable differences even in the alternate one-minute Rest periods.

D. Combined Effects of Alcohol and Sleep Deprivation on Performance, CNS and Autonomic Responses

1. Description

One effect of both alcohol and sleep deprivation is usually considered to be CNS depression. Their combination would be expected to have additive effects, however, common folklore suggests that the effects of these variables may be offsetting (e.g., a very tired person is given a drink to "stimulate" him). The situation is further complicated by the fact that biphasic alcohol effects have been reported for some response systems with low doses having a stimulating effect and high doses having a depressing effect (Carpenter, et al, 1961; Goldberg, 1969; Tong, et al, 1974). Furthermore, in otherwise stressful situation or when highly motivated, individuals seem to overcome (at least temporarily) the detrimental effects of alcohol or sleep deprivation (Frankenhaeuser, et al, 1974).

Alcohol in moderate doses and twenty-four hours of sleep deprivation produce similar effects in many response systems but there are also some differences. For each, the degree of performance impairment tends to be mild rather than extreme, alertness is diminished and the amplitude of EEG components tends to be reduced. But alcohol and sleep deprivation differ with respect to effect on autonomic activity since alcohol is usually reported to increase heart rate but sleep deprivation causes little or no change in heart rate or other autonomic activity. Also, subjects report diminished anxiety following alcohol but increased anxiety following sleep deprivation.

There have been few formal studies of the effects of combinations of alcohol and sleep deprivation, especially studies involving multiple response systems. In the present study we observed the combined effects of one of three doses of alcohol and normal sleep or sleep deprivation on performance, contingent negative variation, visual and auditory evoked potentials, heart rate and subjective feelings of anxiety and alertness. The purpose was to see if similar patterns of change occurred in different response systems and whether the various systems were intercorrelated in their responsiveness.

2. Method

Twenty-four subjects were assigned to one of three alcohol dose groups of 8 subjects each. Each subject received the same dose on two sessions, a normal sleep session and a sleep deprivation session. The doses were .9 ml/kg, .45 ml/kg and 0 ml/kg of 95% ethanol in fruit juice. Mean peak BALs for the .9 and .45 ml/kg doses were .062 and .022%.

For the CNV recordings, silver-silver chloride electrodes were attached to vertex (Cz) and referred to the left ear. The EEG was amplified by a DC amplifier, set to a band pass of 10 Hz. Eye movements and blinks were monitored by electrooculogram.

Two tasks were presented on each session. The first (Categorization) was the task during which CNV and other electrical recordings were made in addition to performance measures. During the second task, complex Choice Reaction Time, only performance measures were taken. The Categorization task required the subject to make a judgment as to whether

a two-attribute stimulus matched the preceding two-attribute stimulus. Four judgments were possible which mapped onto four response keys. A one-sec warning tone, whose onset preceded the visual stimulus by 1200 msec, served as S_1 for the CNV paradigm. The intertrial interval was 11 sec. The task consisted of 120 trials. Motor responses were made only to the visual stimulus (S_2). The CRT task consisted of two parts, a "choice" part and a "keeping-track" part involving short-term memory updating in which the subject had to keep track of whether more right hand or left hand responses had been made and respond accordingly on every 9th trial.

On the night of the sleep deprivation session, subjects spent the night awake in the lab and were monitored by a research assistant. Testing took place the next morning. Eating, drinking and drug use were prohibited except for a 4 A.M. snack. Testing took place at the same time of day for both normal sleep and sleep deprivation sessions.

3. Results

a. Performance

Significant sleep by dose interactions were obtained for error rate and mean RT for the Categorization task. Error rate (Fig. 15) showed a large increase during the sleep deprived-moderate alcohol condition indicating substantial impairment in accuracy for this treatment combination although when each treatment was presented alone, only a small and non-significant effect was found. Reaction time (Fig. 16) showed a different pattern. The effect of moderate alcohol with normal sleep was

to slow RTs ($p < .05$) relative to the placebo condition. The effect of sleep deprivation was to slow RT when no alcohol was given ($p < .05$) but to speed up RT when moderate alcohol was given ($p < .05$). This tendency for the sleep deprived-moderate alcohol combination to speed RTs diminished over blocks.

The two parts of the CRT task were analyzed separately. The choice part resulted in few errors and relatively short RTs with no significant treatment effects except for a marginal dose effect for very long RTs ($p < .08$). In the keeping-track part of the task there was a significantly higher error rate ($p < .05$) after sleep deprivation compared to that seen following normal sleep. No sleep by dose interaction was obtained for either part of the CRT task. The choice part of the task was marginally sensitive to dose but not to sleep condition while the keeping-track part was sensitive to sleep but not dose.

b. Contingent Negative Variation and Evoked Response

Two measures of contingent negative variation (CNV) were defined. (1) Average integrated amplitude consisted of the average voltage during the interval from 450 msec after S_1 to the onset of S_2. (2) Peak CNV was the average voltage during the 50 msec period preceding S_2. Since the temporal parameters of the CRT task made it unsuitable for EP and CNV recordings, these measures were taken only during the Categorization task.

Measure of CNV peak and integrated amplitude were not significantly affected by sleep or by alcohol or their combination.

Three prominent peaks in the evoked potential to S_1 (auditory

Stimulus) were identified visually for each subject with the following mean latencies: 196 msec., 326 msec., 440 msec. These were tentatively labeled as P2, N2 and P3. The peak-to-peak amplitudes for P2-N2 and N2-P3 were measured by hand from the tracing of the averaged evoked response for each block of 60 trials. Multivariate analyses of variance for the three latencies (performed separately for each dose group) indicated that latencies were longer during sleep deprivation than normal sleep for the moderate dose only ($p < .05$). Latencies during low and placebo doses were not consistently affected by sleep deprivation. The two amplitude measureswere not significantly affected by either dose or sleep variables.

c. Heart Rate

Mean heart rate is shown in Fig. 18 for the three doses and two sleep conditions. The difference between sleep conditions was significant only for the moderate dose group and only during the initial block of trials. The direction of the difference was that HR increased with moderate alcohol following normal sleep but decreased with moderate alcohol following sleep deprivation.

d. Alertness

As would be expected, subjects' self-ratings of alertness were lower following sleep deprivation than following normal sleep ($p < .001$). A significant interaction of sleep by dose by phase of session was found ($p < .01$). As shown in Fig. 19, the alertness of the moderate dose group

was less affected by sleep deprivation than the placebo and low dose groups. This tendency became stronger as the session progressed.

e. Anxiety

Self-ratings of anxiety are shown in Fig. 20. The subjects rated themselves more anxious following sleep deprivation than following normal sleep ($p < .05$). This effect also interacted with dose with the increase in anxiety following sleep deprivation occurring only in the placebo and low dose groups. The lack of increased anxiety in the moderate alcohol group suggests that alcohol may have counteracted the anxiety normally induced by sleep deprivation.

f. In summary, sleep by dose interactions were found for most of the measures taken, indicating that the combination of alcohol and sleep deprivation is not a simple additive effect. The only measures not showing a sleep by dose interaction were performance on the CRT task, contingent negative variation (which produced no significant effect) and evoked potential amplitude. Measures which did show sleep by dose interactions were performance on the Categorization task, evoked potential latencies, heart rate, alertness and anxiety. The direction of the interaction was usually that the low dose increased the sleep deprivation trend while the moderate dose attenuated that trend.

4. Comments

When sleep deprivation and a moderate dose of alcohol were combined,

the result was not the addition of the effects of each alone. If the effects of sleep deprivation and moderate alcohol had been additive, their combined effect should have been slow RTs, greatly reduced alertness, very high anxiety and little or no effect on performance accuracy. Rather the combined variables resulted in faster RTs, greater subjective ratings of alertness and lower anxiety than at the other doses while accuracy was severely impaired. This combination of variables also reduced heart rate and altered evoked potential components. The combination of low alcohol and sleep deprivation produced effects similar in direction but less intense than the combination of moderate alcohol and sleep deprivation.

This study has shown that for a variety of measures, the effect of sleep deprivation and moderate alcohol cannot be predicted from knowledge of the effects of each in isolation. Many studies of the interaction of alcohol and stress have attempted to explain the resulting interactions by postulating a unidimensional arousal continuum which is jointly altered by the stress, the particular dose of alcohol and by the subject's pre-existing state. Impairment of performance is thought to occur when arousal is below or above the optimum level for that particular task. While the concept of arousal has been convenient as a post-hoc explanation of alcohol-stress interactions it has proved limited in predictive ability. Although the arousal framework can be applied to certain results of the present study, other of the results require a more complicated model.

When one attempts to examine more closely the ability of the classical arousal conceptual framework to predict results such as these, one becomes aware of complexities in the available data that interfere with

the prediction process. For example, sleep deprived subjects who are at rest show one pattern of physiological response (lower than normal heart rate, respiration rate, muscle tension, skin conductance) while sleep-deprived subjects who are in a situation provoking high anxiety or motivation produce physiological responses that may be abnormally elevated. Such high arousal may reflect a high level of effort needed to maintain normal performance resulting in an increased demand for energy. Thus, following sleep deprivation the subject's physiological indices which form the basis for the definition of his "state of arousal" might be quite different depending on the nature of the task or other activity in which he engages. Since indices of arousal can be influenced by the subject's attempts to stay awake it is difficult to separate the direct effects of sleep deprivation from the secondary effects. As a consequence, predictions which are based on arousal notions must (1) be highly specific with respect to the particular response measure (2) must take into consideration the total constellation of responses which have just occurred, and (3) must consider knowledge of possible biphasic action for each response system to each stressor.

CONCLUSIONS

The results of this research form a highly intricate and somewhat confusing picture, but a few conclusions can still be drawn. The clearest result is that the effect of moderate to high doses of alcohol on the EEG was to reduce and slow beta 1, to slow alpha, to increase and speed theta and to slow delta. The pattern is that of a deactivation of the CNS.

With respect to the relationship between the performance and the EEG, there was no reliable association at low and moderate levels of alcohol (Study A), although large performance differences were found. This finding suggests that, in this relatively benign experimental situation, some CNS process other than that represented by the EEG must be associated with fast and slow performance.

At high levels of alcohol (Study B) and under situations of high motivation (Study C), there appeared to be an association between certain EEG parameters and the performance measure. This finding suggests that when the system is stressed by requiring it to perform under adverse circumstances (when highly intoxicated) or when highly motivated then the EEG becomes relevant.

The nature of the relationship between performance and EEG in Studies B and C varies somewhat with the different experimental circumstances. Common to both studies is the finding that, irrespective of BAL, fast performance is related to reduced beta 1 power. Furthermore, in Study B, at high BALS, fast performance is related to faster alpha, implying that in order to respond quickly when highly intoxicated, faster alpha frequency is required. A different pattern was seen in

Study C. There, at high BALS, fast performance was associated with more beta 1 power. Both of these findings (faster alpha and more beta 1) are indicative of an activated CNS. Thus, we see a rather paradoxical situation where, when BAL is not segregated, fast performance is related to reduced beta 1 power. The beta 1 "generator" seems to get turned up or down depending on the state of the individual in order to yield an output of consistently fast performance. Theta was also implicated in both studies but not in a consistent way.

The incentive condition (Study C), irrespective of BAL or performance, had an activating effect as indicated by faster alpha and more and faster beta 1. When the incentive condition was paired with high BALs (irrespective of performance) the effect was faster alpha and more beta 1 (again indicating activation) and reduced and slower theta.

When fast performance occurred in the incentive condition the effect differed for Performance and Rest minutes. The only effect during the Performance minutes was slower delta (suggestive of changes in eye movements). During Rest, fast theta and slow alpha were seen, possibly related to the processing of the feedback information regarding whether or not an incentive had been earned (this information was provided the subject immediately after each block of performance).

Changes in beta 2 power and moments have not been interpreted because of the possibility that this band is contaminated by muscle artifact. This view is supported by noting that beta 2 increased at high BALs when the subjects were inclined to fidget.

EEG data was not available for Study D but the other physiological and performance measures indicated that the combination of stress (in the form of sleep deprivation) and alcohol produced interaction effects.

The low dose of alcohol tended to increase the sleep deprivation trend while the moderate dose attenuated the trend.

In this series of studies, there was no consistent evidence of clearly differentiated EEG states related to fast and slow performance other than finding more CNS activation when fast performance occurred with high alcohol. Without the state of high alcohol, fast performance was related to decreased CNS activation. Thus the systems that are related to fast performance seem quite sensitive to the amount of activation. CNS activation showed an increase overall during the incentive condition but was not differentiated for periods of fast and slow performance unless high alcohol was also a factor.

From these data it seems that the best strategy for looking for EEG-performance relationships is to put a load on the system either in the form of a handicap (high alcohol) or stress. To look for CNS correlates of good and bad performance when the system is in normal condition would seem to require measures other than scalp EEG.

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TABLE 1

EEG Variables Whose Partial Correlation With Blood Alcohol Level or Performance Reached Significance at $p < .001$ for at Least Two Out of Four Channels. (+) Indicates Positive Correlation.

PARTIAL CORRELATION WITH BAL

S #		Delta		Theta		Alpha or Alpha-Betal		Beta 1		Beta 2	
		Pow	Mom	Pow	Mom	Pow	Mom	Pow	Mom	Pow	Mom
1	Perf	-		+	+	-			-	-	+
	Rest			+	+	-	-		-	-	+
2	Perf		-		+		-	+		+	-
	Rest		-	+	+		-		-		
3	Perf					-	+	-			
	Rest							-	-	+	+
4	Perf		-	+	+	+			-	-	-
	Rest	+	-	+	+	+	-		-	-	-
5	Perf	-		-	+	+			-		
	Rest			-	+		-		-		

PARTIAL CORRELATION WITH PERFORMANCE

S #		Delta		Theta		Alpha or Alpha-Betal		Beta 1		Beta 2	
		Pow	Mom	Pow	Mom	Pow	Mom	Pow	Mom	Pow	Mom
1	Perf					+	+		-		
	Rest										
2	Perf						-				
	Rest						-				
3	Perf										
	Rest										
4	Perf										
	Rest										
5	Perf										
	Rest										

Table 2

Multiple Correlations R of BAL with EEG variables
(all F significant at p <.01)

Subject	R	R^2	D.F.	F	F (p <.01)
1	.6665	.4442	33/722	17.49	1.74
2	.7430	.5520	40/579	17.83	1.63
3	.5291	.2800	33/594	7.00	1.74
4	.8005	.6408	33/633	34.22	1.74
5	.7586	.5754	32/402	17.03	1.74

Table 3

Multiple Correlations R of Performance with BAL (Step 1) and with EEG variables (Step 2)

Subject	Step	R	R^2	D.F.	F	F ($p < .01$)	$p < .01$
1	1	.3350	.1122	1/754	95.33	6.69	yes
	2	.5426	.2944	37/718	8.095	1.74	yes
2	1	.0368	.0014	1/618	0.838	6.69	no
	2	.3500	.1225	41/578	1.968	1.63	yes
3	1	.0214	.0005	1/626	0.287	6.69	no
	2	.2533	.0642	37/590	1.093	1.74	no
4	1	.2240	.0501	1/665	35.12	6.69	yes
	2	.3543	.1255	37/629	2.44	1.74	yes
5	1	.1674	.0281	1/433	12.48	6.72	yes
	2	.3821	.1460	33/401	2.08	1.74	yes

TABLE 4a

SUMMARY OF SIGNIFICANT TRENDS OF EEG PARAMETERS VS PERFORMANCE AT EACH BLOOD ALCOHOL LEVEL

LINEAR TRENDS

BAL = 0

S #	Delta		Theta		Alpha		Beta 1		Beta 2	
	Pow	Mom	Pow	Mom	Pow	Mom	Pow	Mom	Pow	Mom
1			-	-	-	-		+	-	
2										
3										
4			+			+		+		
5				+		+				-

BAL = LOW

1	+	+							-	-
2	+	-				+		-		
3										
4										
5										

BAL = MODERATE

1			+	-						
2										
3										
4				+						
5										

BAL = HIGH

1	-			-					+	
2	+			-		+	+		+	
3										
4					+	+				-
5				-				-		

TABLE 4b

QUADRATIC TRENDS

BAL = 0

S #	Delta		Theta		Alpha		Beta 1		Beta 2	
	Pow	Mom	Pow	Mom	Pow	Mom	Pow	Mom	Pow	Mom
1			-	-		-		-	-	
2										
3										
4			-					-		
5										

BAL = LOW

1						
2						
3						
4						
5						

BAL = MODERATE

1						
2						
3						
4						
5						

BAL = HIGH

1	-	+	+	+		+		
2								
3	+							
4			-			-		
5								

TABLE 4c

CUBIC TRENDS

BAL = 0

S #	Delta		Theta		Alpha		Beta 1		Beta 2	
	Pow	Mom	Pow	Mom	Pow	Mom	Pow	Mom	Pow	Mom
1										
2	+	-		-		+				
3										
4										
5										

BAL = LOW

1	+	+				
2						
3						
4						
5						

BAL = MODERATE

1		-		-		
2			-		-	-
3						
4						
5						

BAL = HIGH

1						
2						
3						
4						
5				-		

Table 5

Dose, BAL, intoxication self-rating and RT difference scores (alcohol minus placebo) for each subject.

	Session	Total Dose (ml)	Mean BAL	Self Rating	Mean RT Difference Scores Undiv.	t	p	Div.	t	p
Subj. #1	1	96	.069	4.71	50.7	4.40	<.01	89.1	5.13	<.01
	2	72	.055	2.93	7.9	1.00	ns	24.9	1.59	ns
	3	80	.054	2.46	5.2	0.60	ns	35.9	3.90	<.01
	4	80	.034	4.14	-0.7	0.10	ns	13.9	1.43	ns
	5	104	.054	4.07	0.7	0.08	ns	18.2	1.73	ns
	6	96	.071	5.25	16.8	1.20	ns	26.4	1.67	ns
Subj. #2	1	88	.058	6.43	54.0	3.90	<.01	63.6	3.36	<.01
	2	110	.086	7.18	30.0	2.12	<.05	20.0	1.30	ns
	3	88	.076	7.07	10.0	0.94	ns	22.1	1.50	ns
	4	100	.062	5.54	18.0	1.80	<.05	24.6	1.22	ns
	5	77	.053	5.54	24.0	2.59	<.05	26.1	1.10	ns
	6	88	.060	5.57	9.0	0.95	ns	56.4	3.67	<.01
Subj. #3	1	90	.104	4.75	39.9	4.57	<.01	85.4	3.22	<.01
	2	75	.080	5.07	28.4	3.19	<.01	36.8	2.74	<.01
	3	67	.078	4.64	29.1	5.07	<.01	16.8	1.04	ns
	4	67	.070	3.64	26.3	3.71	<.01	63.5	3.24	<.01
	5	45	.046	3.29	21.9	4.00	<.01	3.6	0.40	ns
	6	60	.060	3.64	-7.1	0.95	ns	-6.8	-1.01	ns
Subj. #4	1	96	.074	5.61	69.3	3.66	<.01	136.4	4.41	<.01
	2	96	.091	6.61	50.0	2.79	<.01	114.3	5.13	<.01
	3	96	.027	5.21	2.9	0.24	ns	28.6	2.06	<.05
	4	120	.070	4.64	49.3	1.81	<.05	33.0	1.80	<.05
	5	120	.079	4.89	60.7	3.86	<.01	52.6	2.40	<.05
	6	144	.053	4.64	38.2	3.35	<.01	-2.1	0.10	ns
Subj #5	1	132	.094	6.18	34.2	2.63	<.05	54.6	3.00	<.01
	2	88	.069	5.32	44.6	2.68	<.01	66.8	2.71	<.01
	3	99	.053	4.43	15.4	1.15	ns	0.4	0.02	ns
	4	77	.060	4.36	20.0	2.04	<.05	26.1	1.90	<.05
	5	99	.066	4.57	10.0	0.77	ns	52.5	3.64	<.01
	6	110	.063	5.04	27.1	1.80	<.05	48.8	2.30	<.05

Table 6

Mean RT difference scores for undivided and divided tasks and t-tests of their difference.

Subject	RT Difference Scores		t	p
	Undivided	Divided		
1	13.43	34.90	4.78	<.01
2	24.16	35.46	1.43	ns
3	23.08	33.21	0.95	ns
4	45.06	60.41	0.84	ns
5	25.20	41.53	2.07	<.10

Table 7

Intercorrelations and multiple correlations of mean BAL, mean self-rating of intoxication and composite RT difference score (alcohol minus placebo).

S #1

	Mean BAL	Mean Rating	Composite RT Difference Score
Session	-.04	.31	.41
Mean BAL		.56	.76
Mean Rating			.24
Multiple R (Sessions + BAL)			.85*
Multiple R (Rating + BAL)			.63

S #2

	Mean BAL	Mean Rating	Composite RT Difference Score
Session	-.31	-.64	-.13
Mean BAL		.70	-.50
Mean Rating			.01
Multiple R (Sessions + BAL)			.58
Multiple R (Rating + BAL)			.50

S #3

	Mean BAL	Mean Rating	Composite RT Difference Score
Session	-.94*	-.90*	-.83*
Mean BAL		.93*	.77
Mean Rating			.56
Multiple R (Sessions + BAL)			.83*
Multiple R (Rating + BAL)			.77

S #4

	Mean BAL	Mean Rating	Composite RT Difference Score
Session	-.31	-.84*	-.60
Mean BAL		.53	.83*
Mean Rating			.59
Multiple R (Sessions + BAL)			.90*
Multiple R (Rating + BAL)			.72

S # 5

	Mean BAL	Mean Rating	Composite RT Difference Score
Session	-.49	-.49	-.37
Mean BAL		-.89*	.88*
Mean Rating			.88*
Multiple R (Sessions + BAL)			.88*
Multiple R (Rating + BAL)			.82

*p <.05 (one tail)

Table 8

ANALYSIS OF VARIANCE FOR STUDY B
(N=5632 One-Second Power Spectral Averages)

		DELTA		THETA		ALPHA		BETA 1		BETA 2	
		Pow	Mom	Pow	Mom	Pow	Mom	Pow	Mom	Pow	Mom
BAL	Perf		-	+	+			-	-		
	Rest			+	+	-	-	-	-		+
Performance	Perf			+				-			
	Rest							-			
BAL X Perf.	Perf						+				
	Rest						+				
F - P	Perf	+	-	-	-	-	-	-			+
	Rest	+	-	-	-	-	-		+		
F-P X BAL	Perf			-							
	Rest			-		+	+				-
F-P X BAL X Perf.	Perf										
	Rest		-				-				
$F_3 - F_4$	Perf			+							
	Rest										
$P_3 - P_4$	Perf							-			-
	Rest							-			-

Table 9

ANALYSIS OF VARIANCE FOR STUDY C
(N=15,378 One-Half-Second Power Spectral Averages)

	DELTA Pow	DELTA Mom	THETA Mom	THETA-ALPHA Pow	ALPHA Mom	BETA 1 Pow	BETA 1 Mom	BETA 2 Pow	BETA 2 Mom	
BAL	-	-	+		-	-	-	+	+	Perf
		-	+	+	-	-	-	+	+	Rest
Perf.			-			-				Perf
	-				+	-	+			Rest
BAL X Perf.			-			+			-	Perf
						+				Rest
INC		-	-		+	+	+		-	Perf
				-		+	+	+		Rest
INC X BAL			-	-	+	+				Perf
			-	-	+	+	+			Rest
INC X Perf.		-							-	Perf
	-		+		-					Rest
F-P	+	-	-	-		-	+	+	+	Perf
	+	-	-	-	+	-	+	+	+	Rest
F-P X BAL		+					+	+		Perf
					+	+	+	+		Rest
F-P X Perf.		-								Perf
										Rest
F-P X INC		+								Perf
		-								Rest
$F_3 - F_4$		-					+	+		Perf
							+	+	+	Rest
F_3-F_4 X INC								+		Perf
								+		Rest
$P_3 - P_4$									-	Perf
										Rest

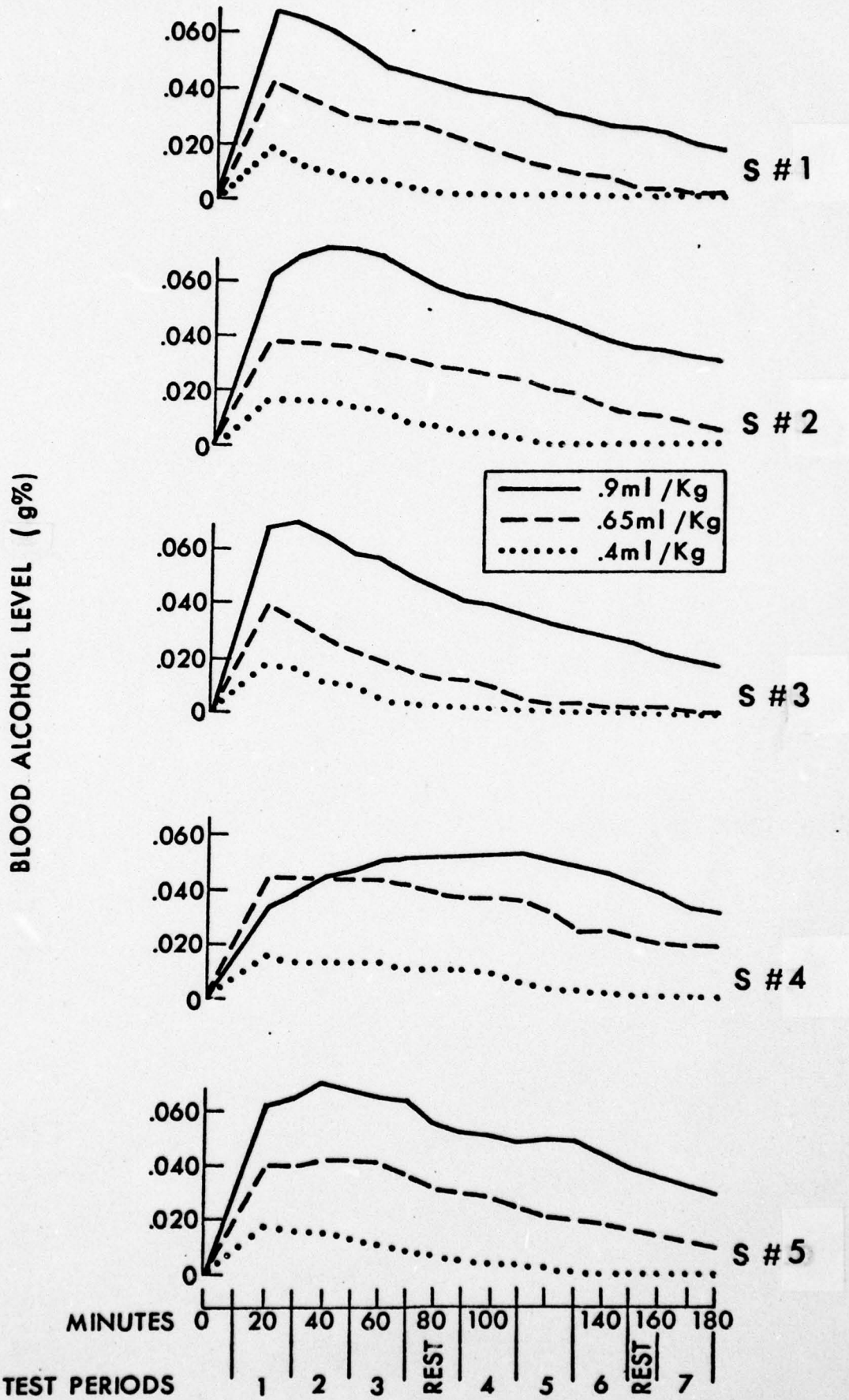


Figure 1. Study A. Time course of blood alcohol level for each dose.

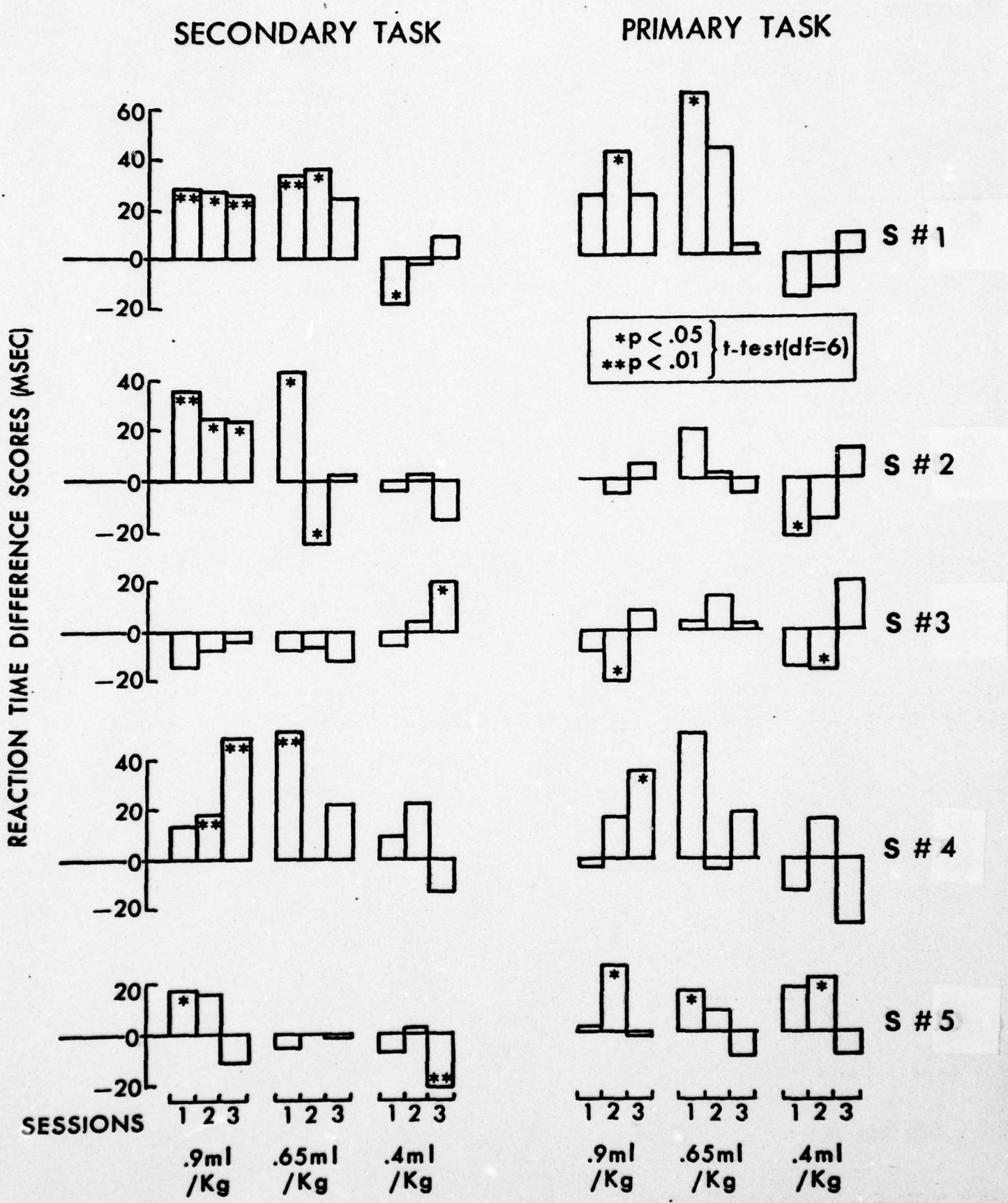


Figure 2. Study A. Reaction time difference scores by dose and session for divided attention tasks.

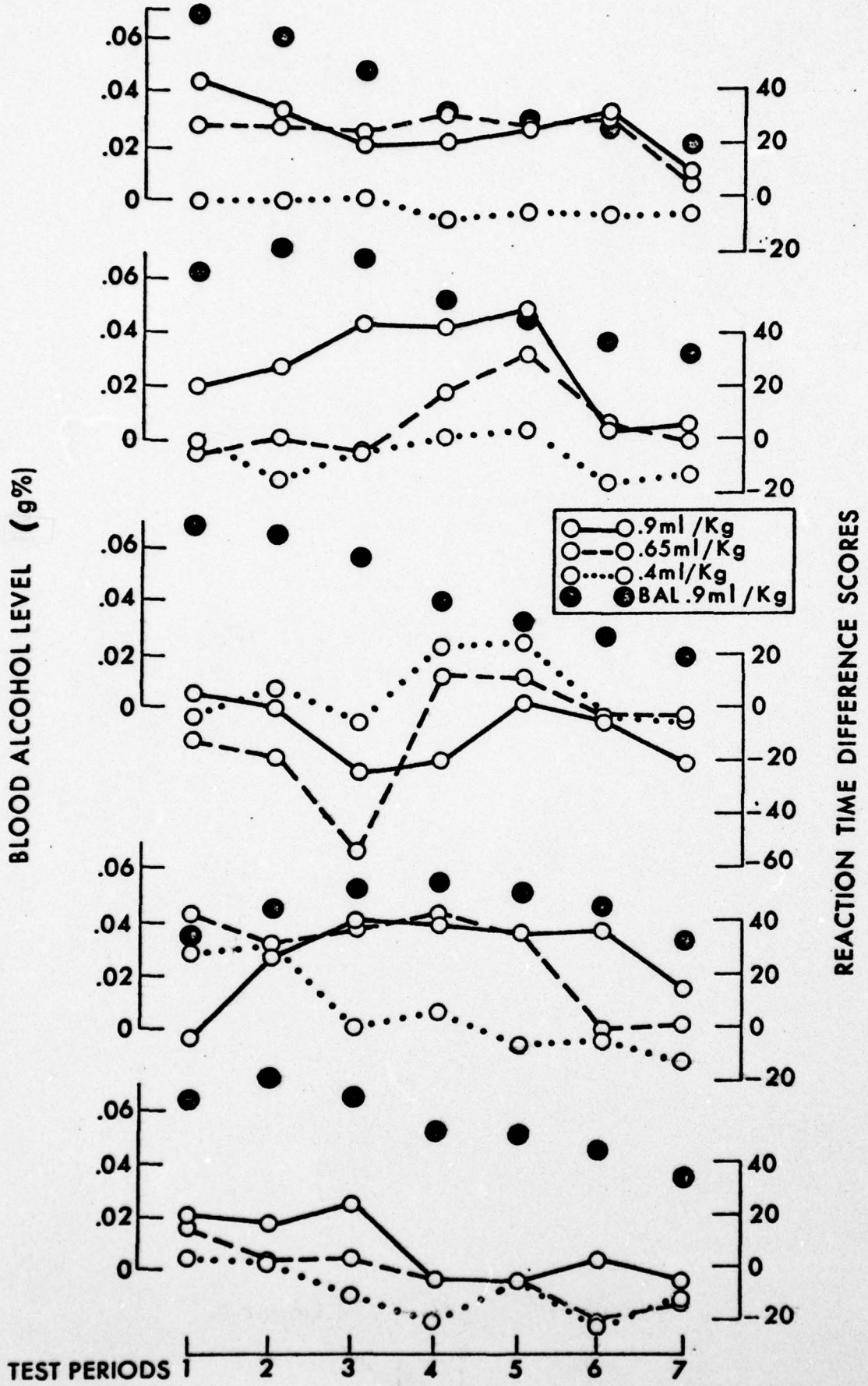


Figure 3. Study A. Time course of reaction time difference scores and blood alcohol levels.

Figure 4. Study A. Factor scores for EEG frequencies on four power spectral factors for subject 1.

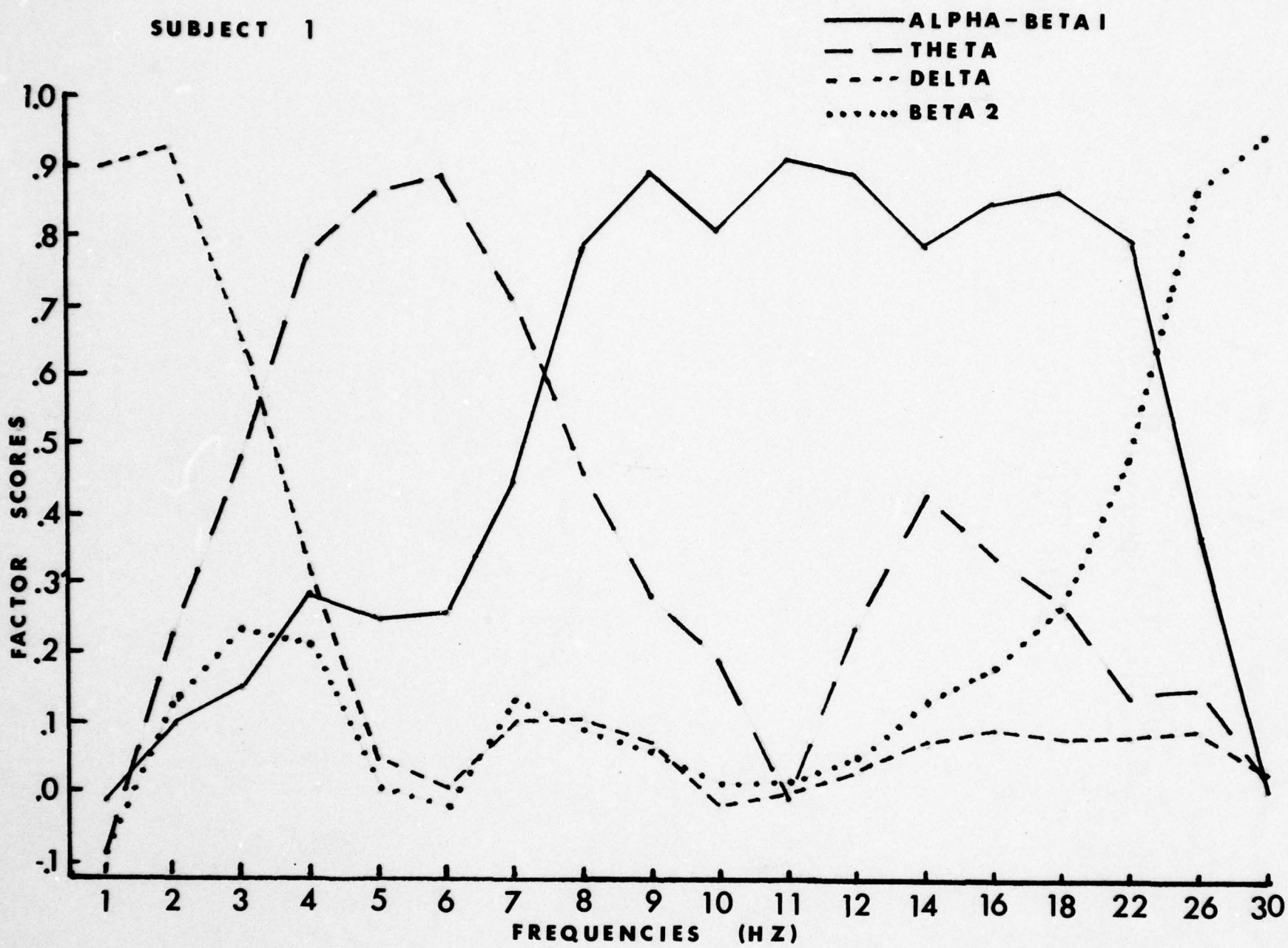


Figure 5. Study A. Factor scores for EEG frequencies on four power spectral factors for Subject 3.

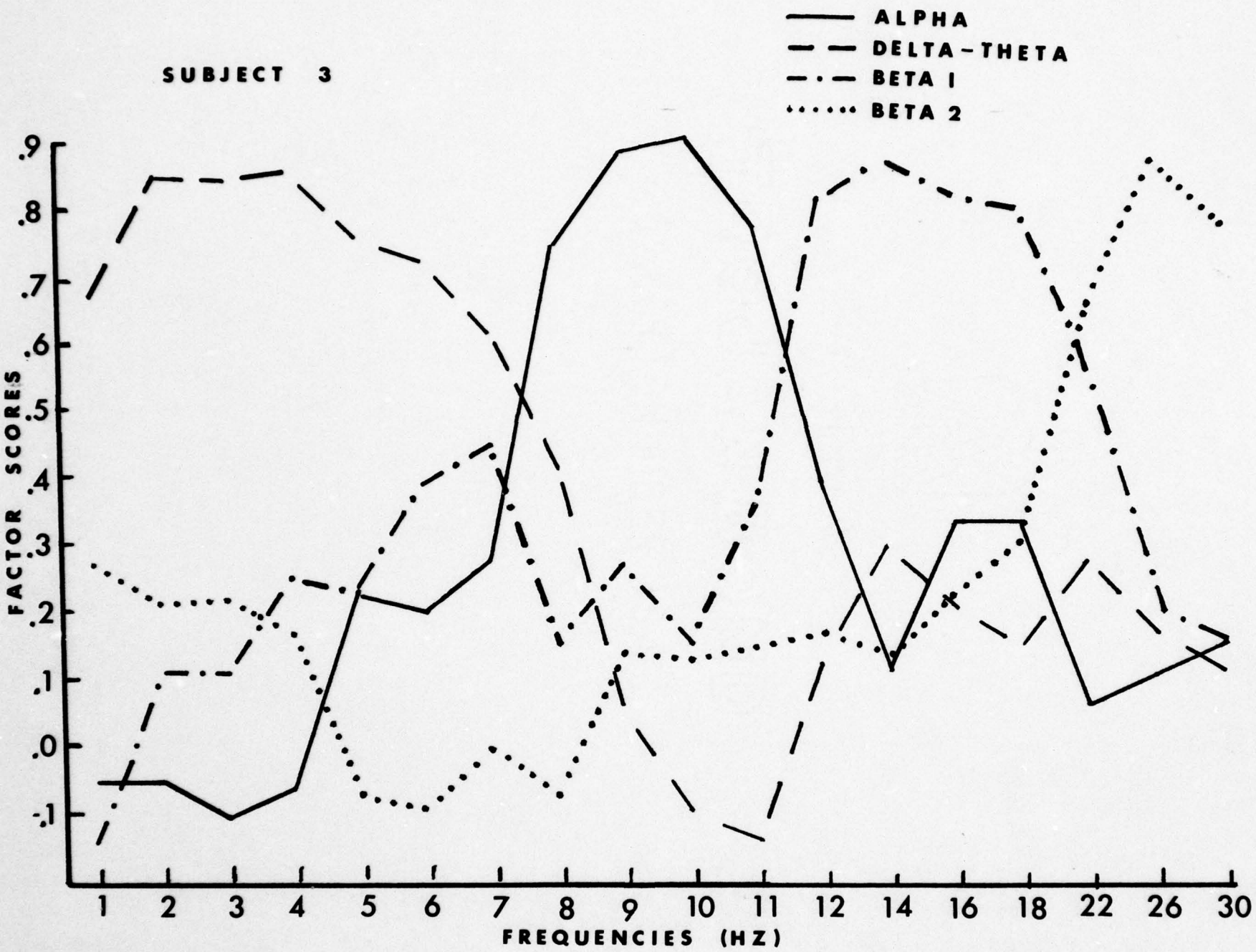


Figure 6. Study A. Factor scores for EEG frequencies on four power spectral factors for Subject 4.

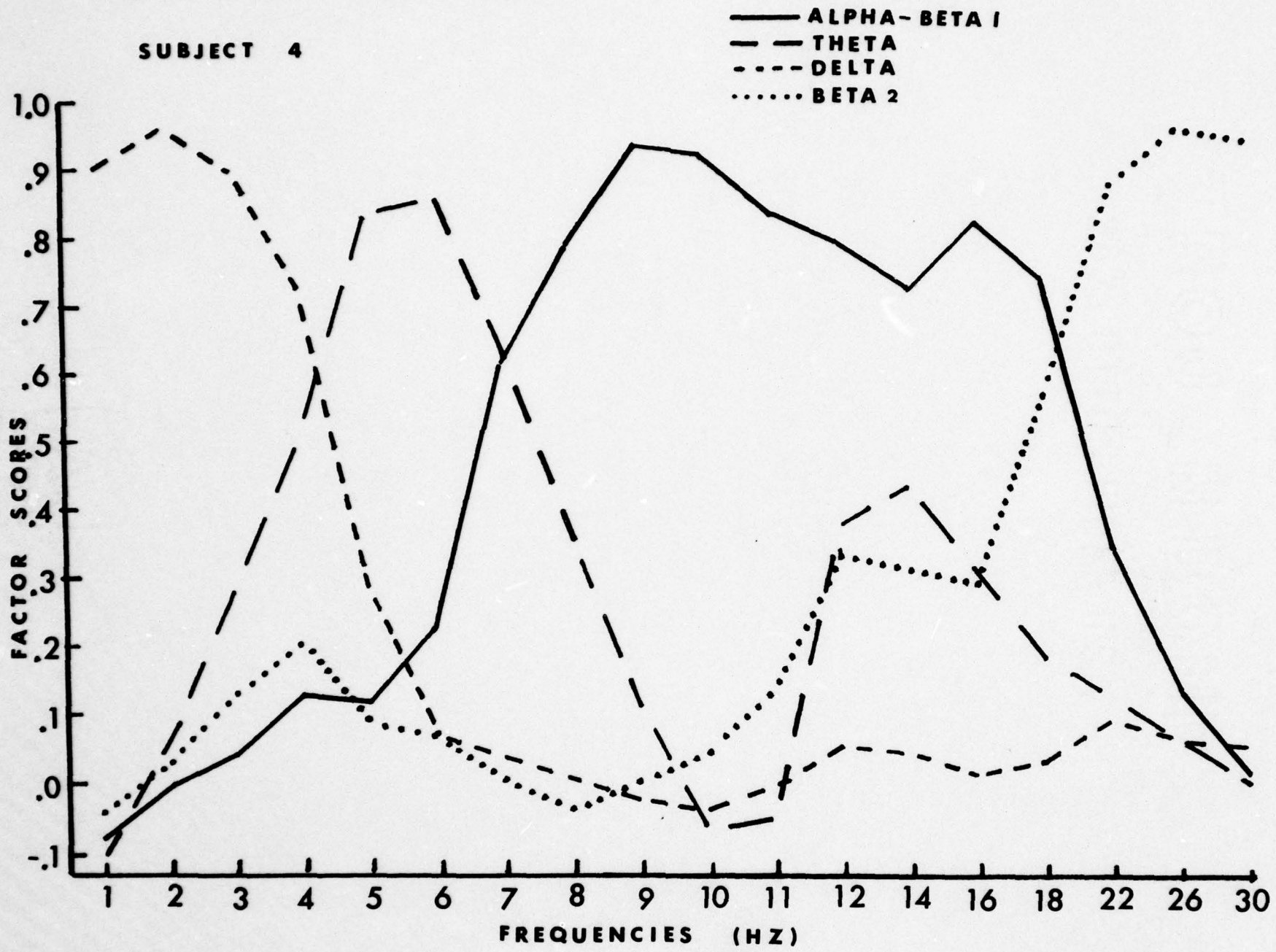


Figure 7. Study A. Factor scores for EEG frequencies on three power spectral factors for subject 5.

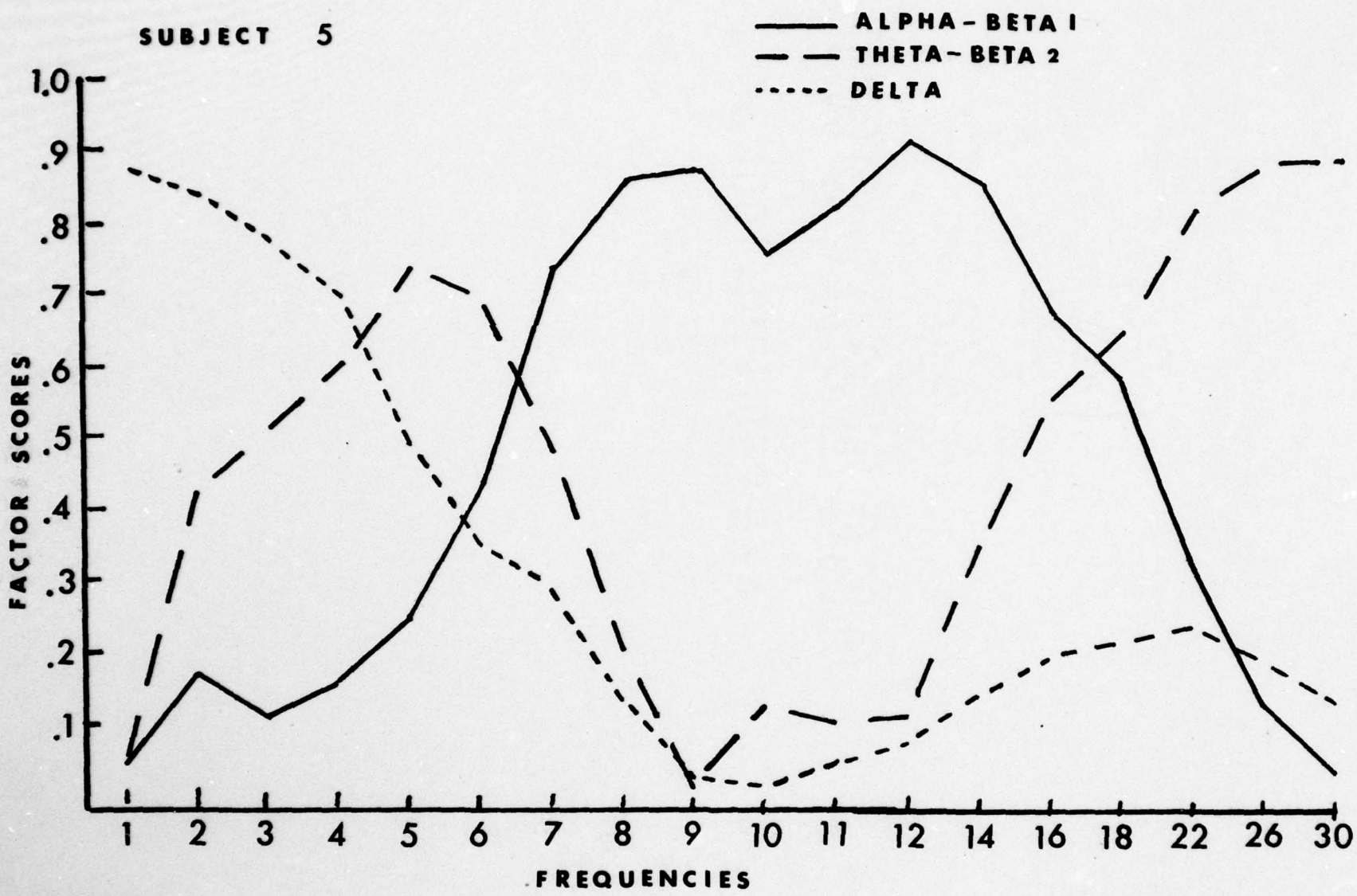


Figure 8. Study B. Factor Scores for EEG frequencies on four power spectral factors for all subjects.

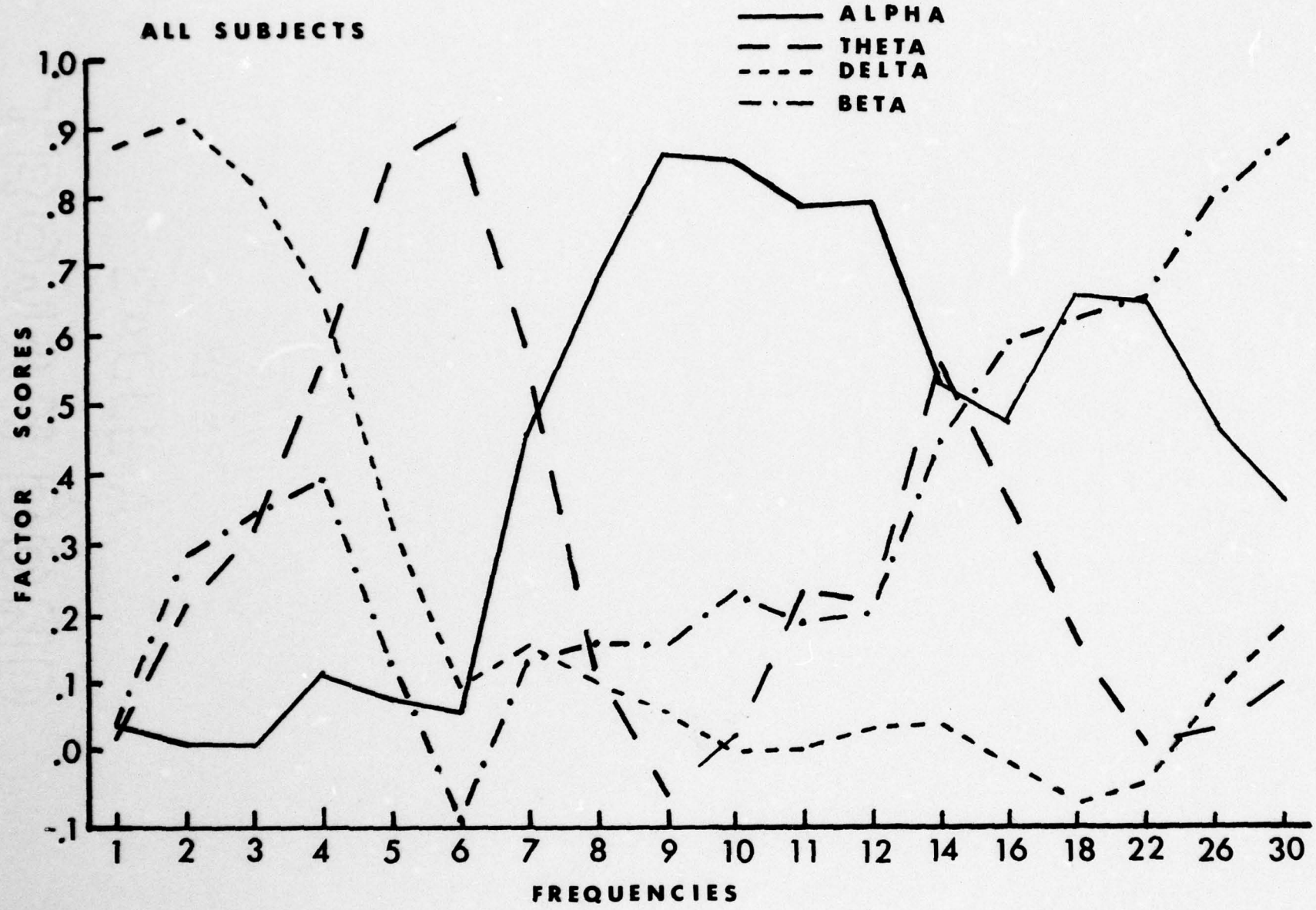


Figure 9. Study C. Mean reaction time for alcohol (A) vs placebo (P) on incentive (I) and no incentive (NO I) sessions for the first and last five blocks.

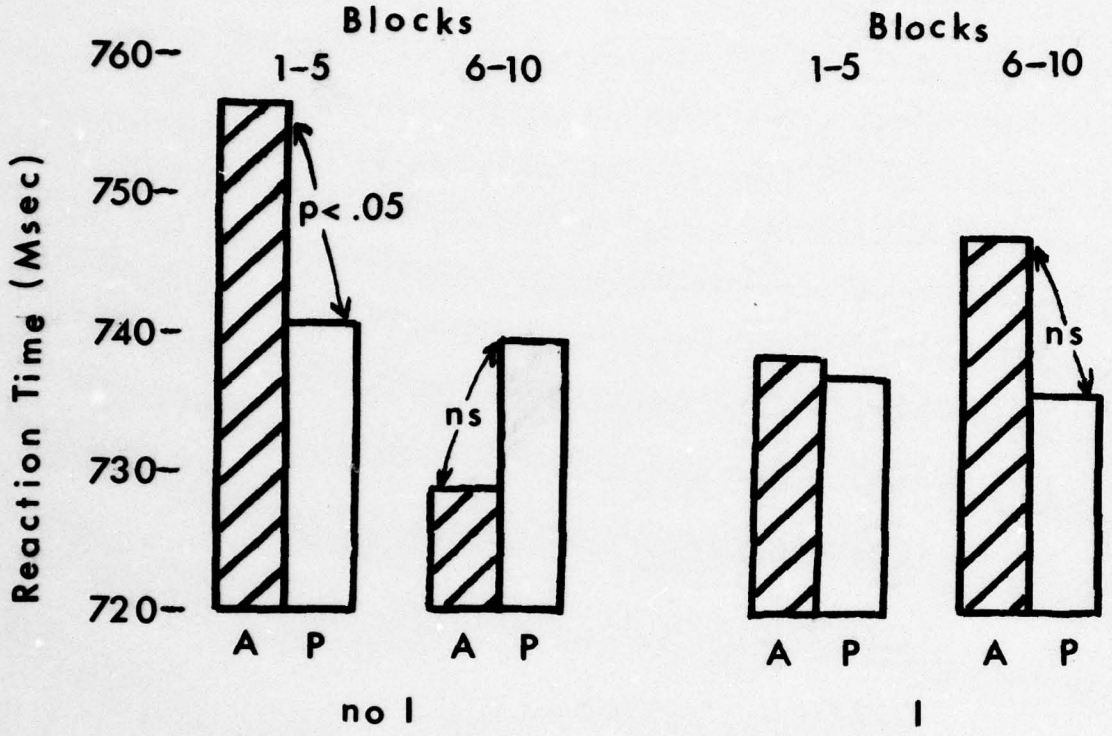


Figure 10. Study C. Number of errors for alcohol (A) vs placebo (P) on incentive (I) and no incentive (NO I) sessions for the first and last five blocks.

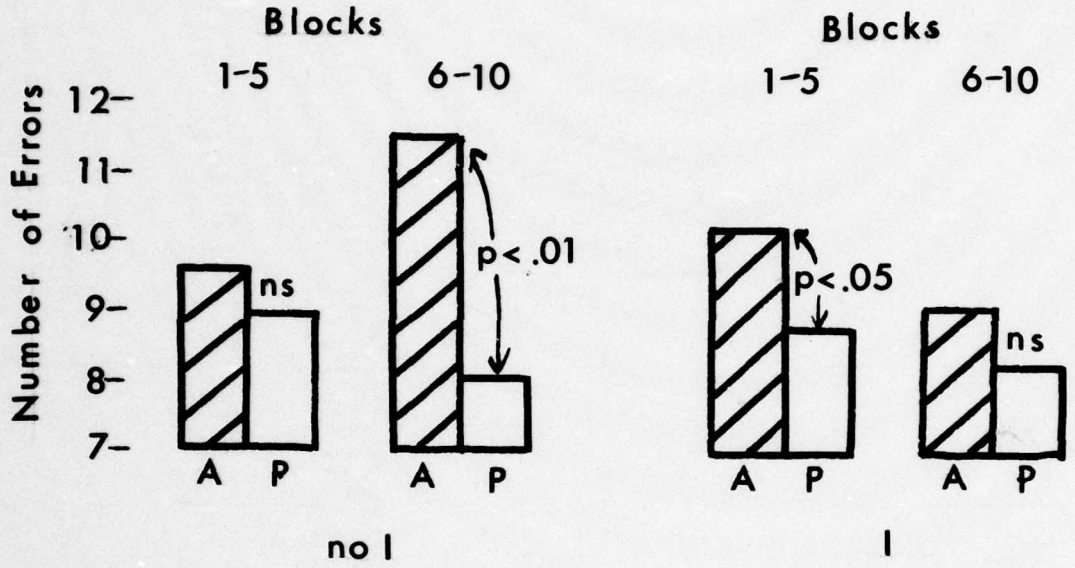


Figure 11. Study C. Mean reaction time and blood alcohol level by blocks for the Alcohol/No Incentive and Alcohol/Incentive sessions.

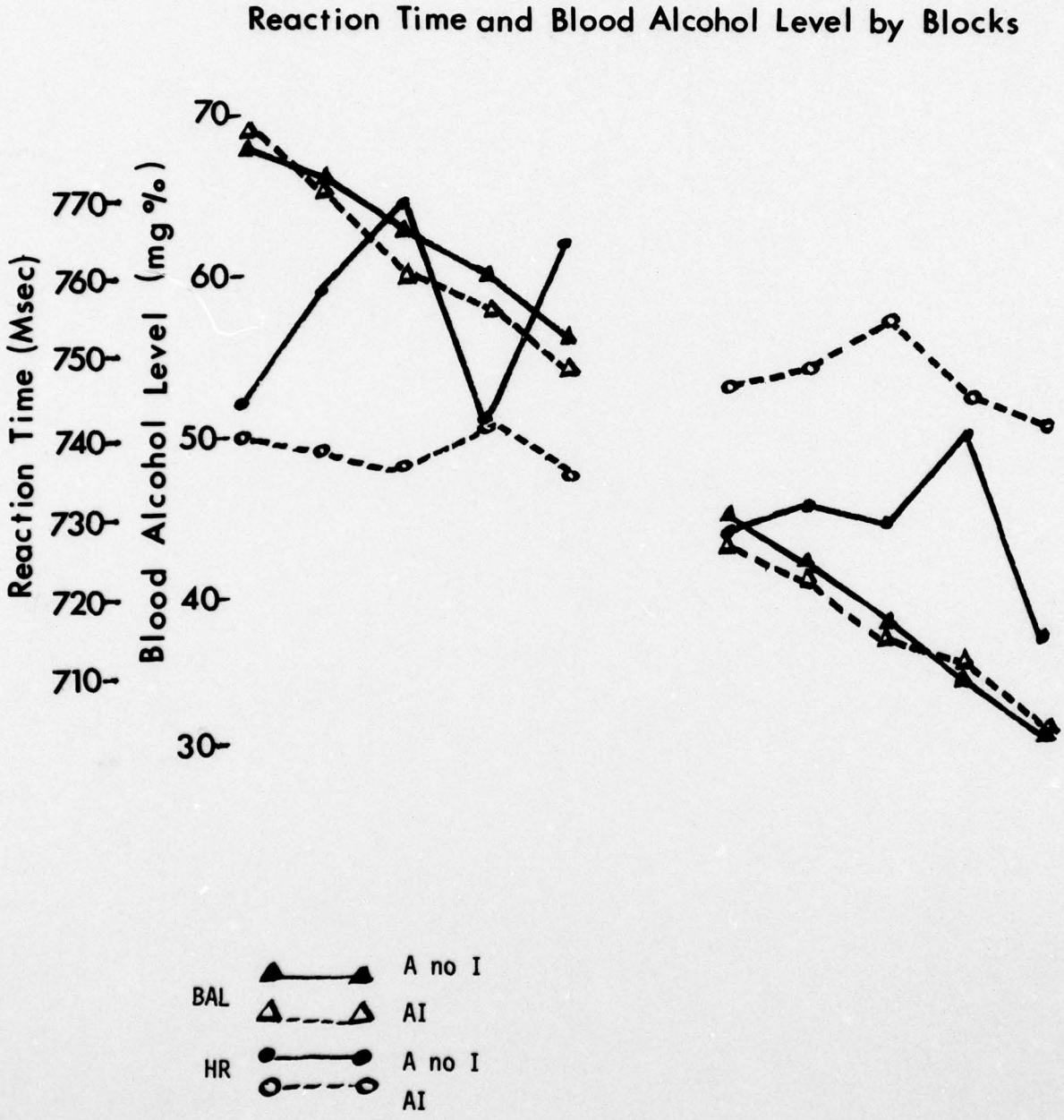


Figure 12. Study C. Mean heart rate for alcohol vs placebo on incentive (I) and no incentive (NO I) sessions for the subjects of sub-groups A (TR, LU, HO) and B (RE, BA, WI).

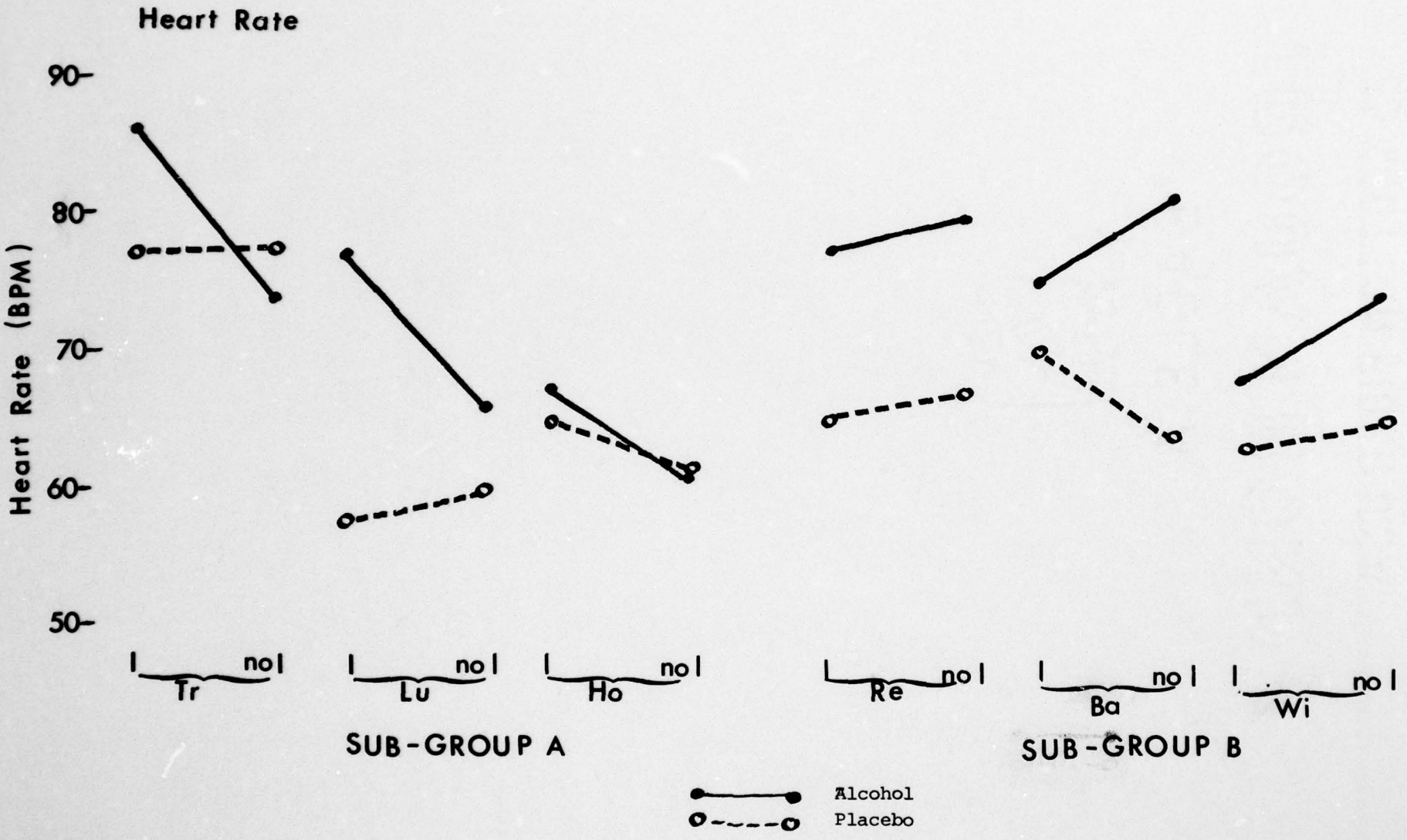


Figure 13. Study C. Mean heart rate and blood alcohol by blocks for the Alcohol/ Incentive and Alcohol/No Incentive sessions.

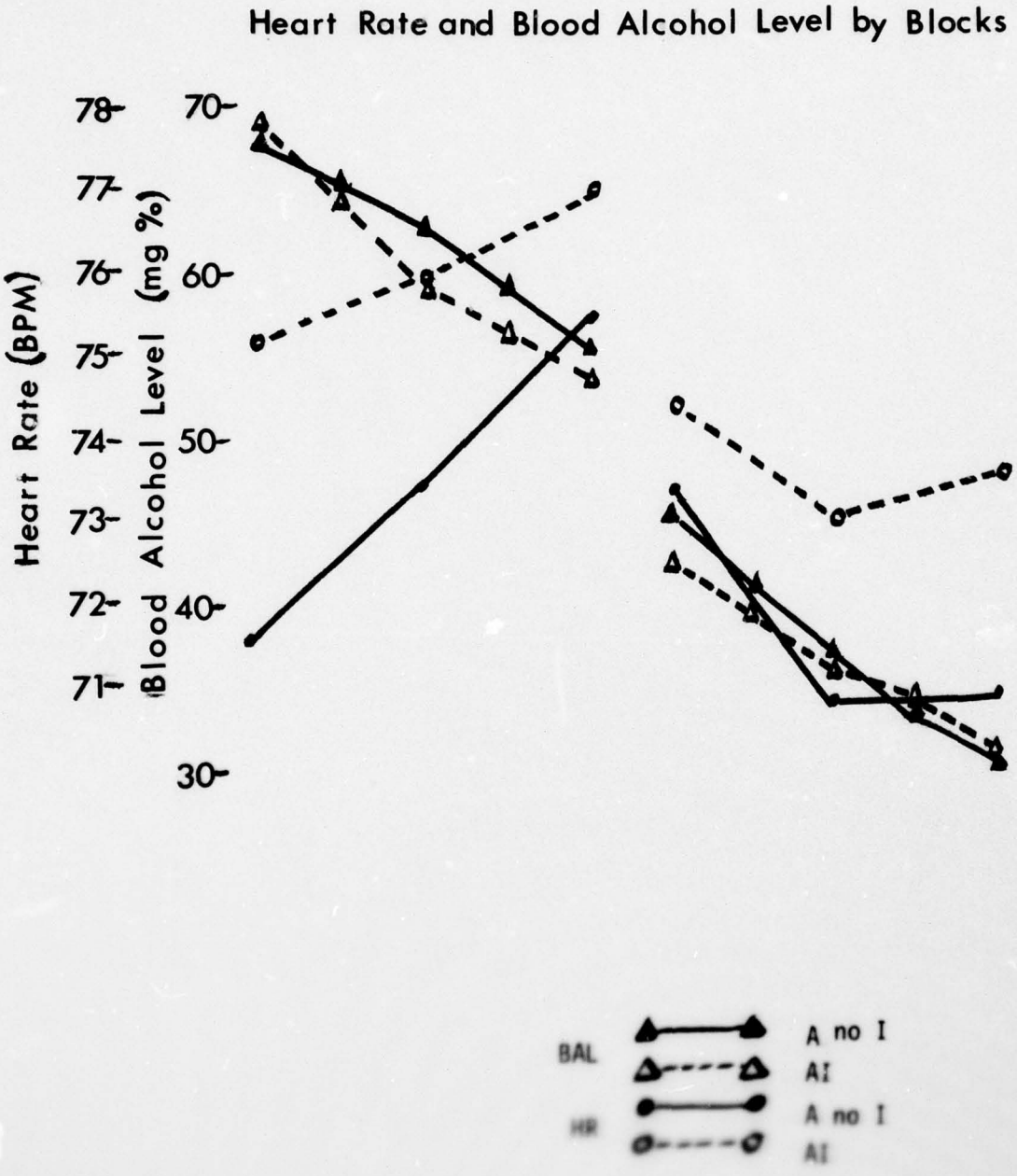


Figure 14. Study C. Factor scores for EEG frequencies on four power spectral factors for all subjects.

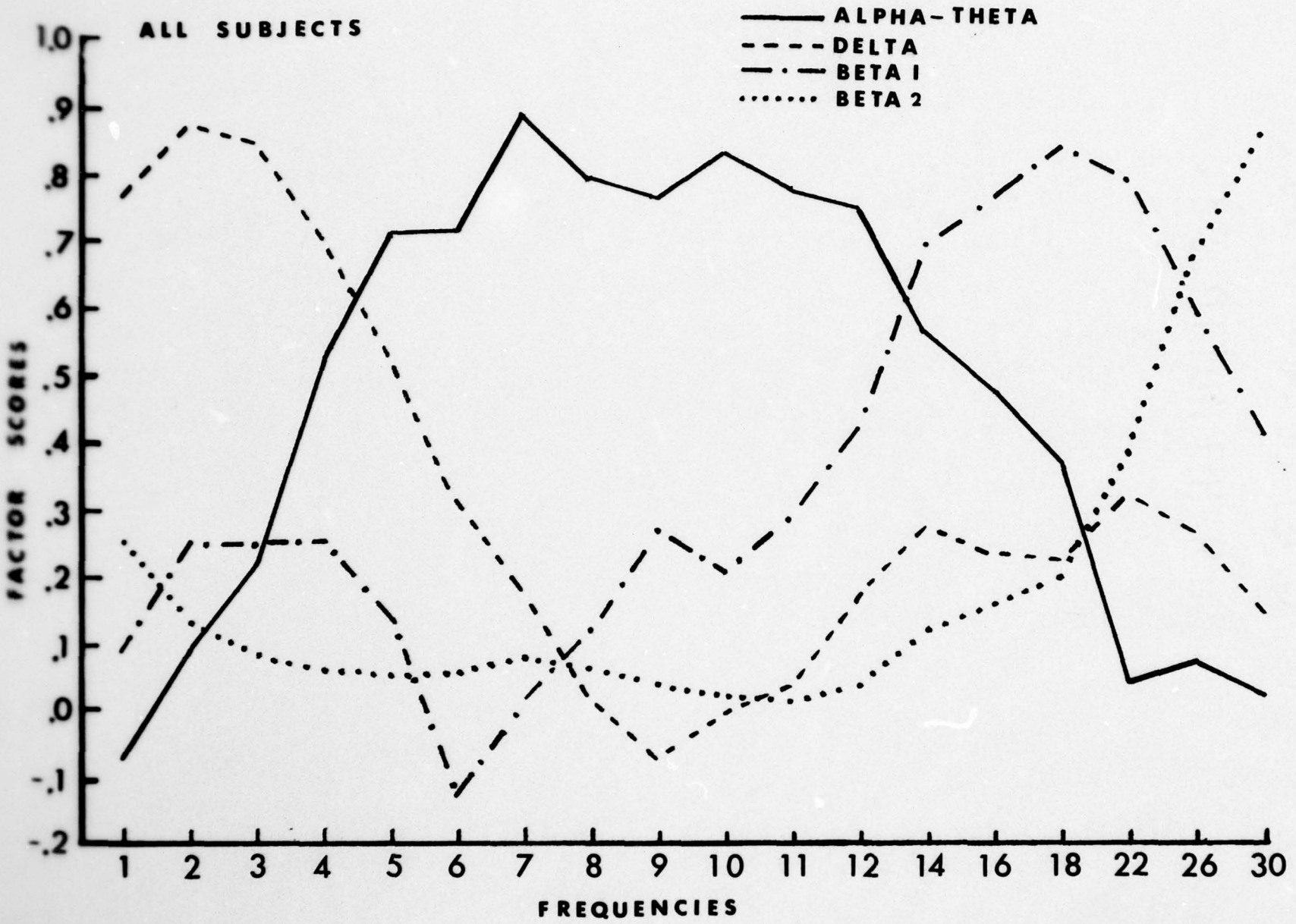


Figure 15. Study D. Mean proportion of errors for normal sleep vs sleep deprived conditions at each alcohol dose.

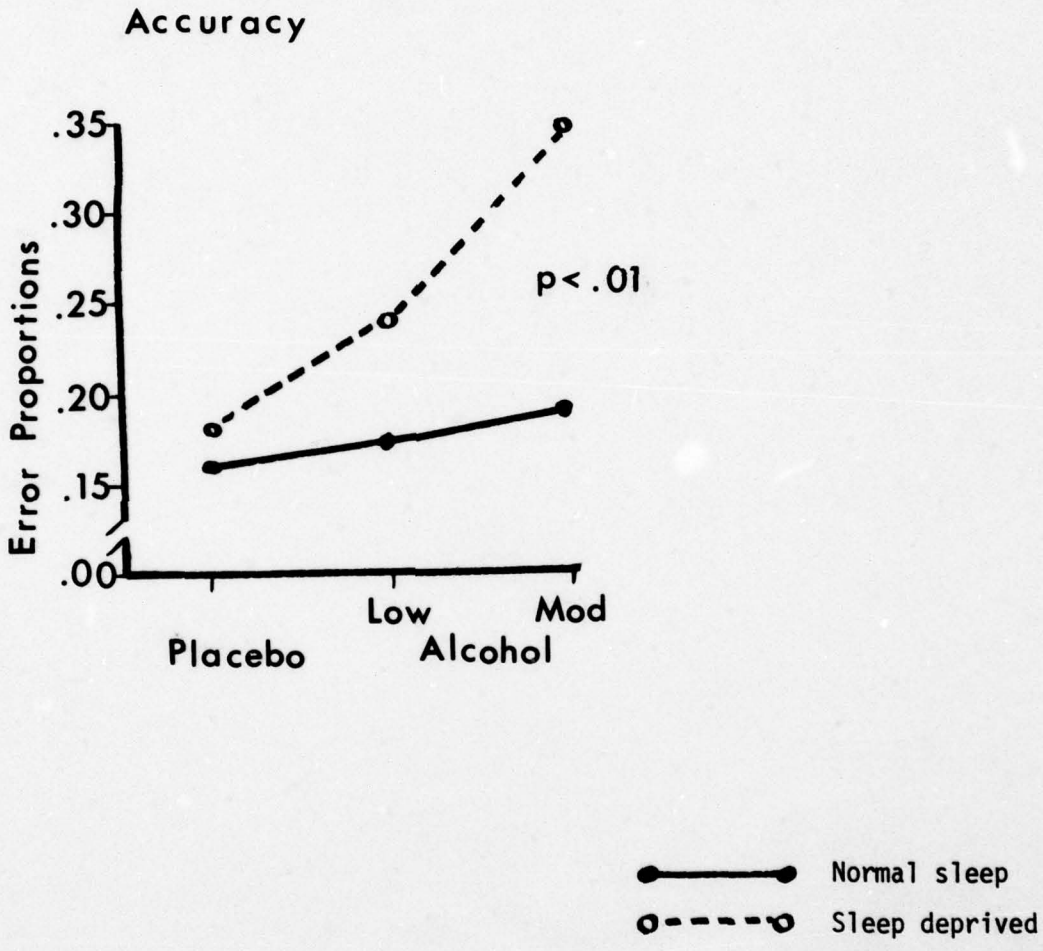


Figure 16. Study D. Mean reaction time for normal sleep vs sleep deprived conditions for each alcohol dose.

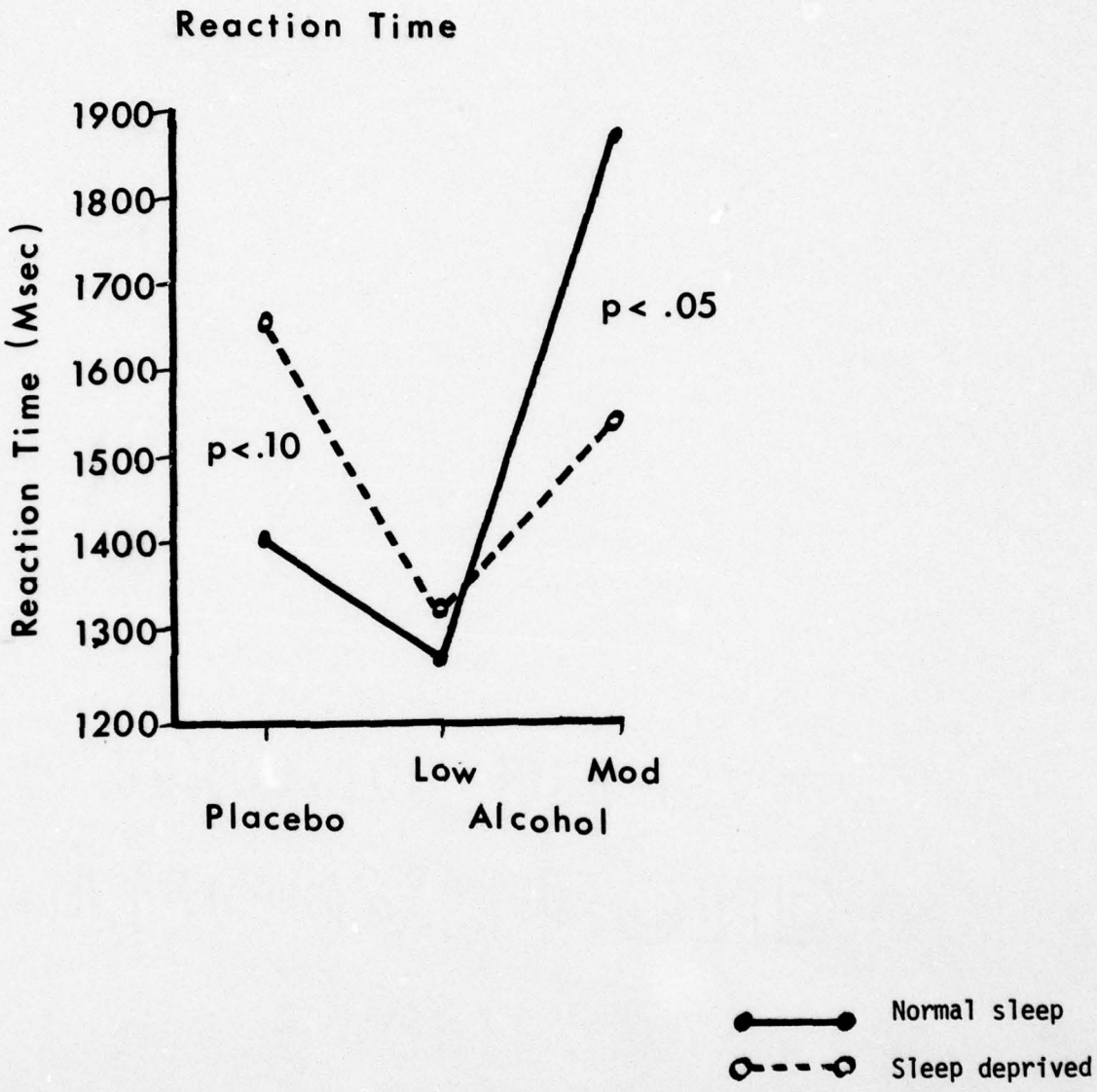


Figure 17. Study D. Evoked potential latency difference scores (sleep deprivation minus normal sleep) to S_1 for each alcohol dose.

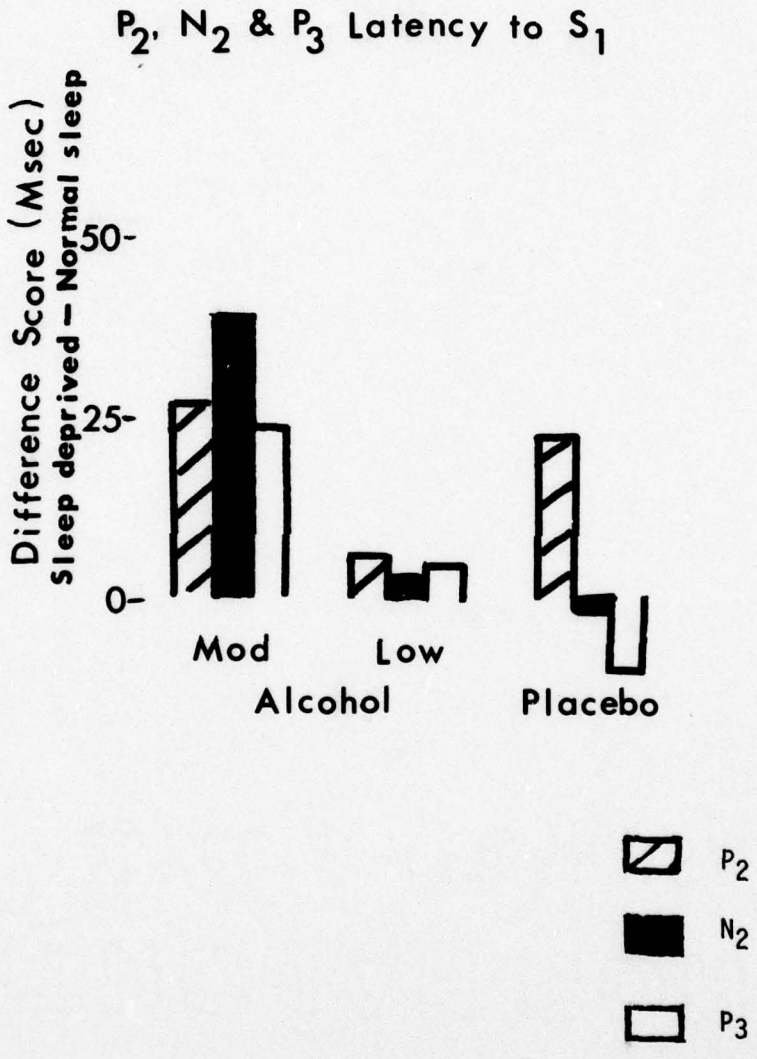


Figure 18. Study D. Mean heart rate for normal sleep vs sleep deprived conditions for each alcohol dose.

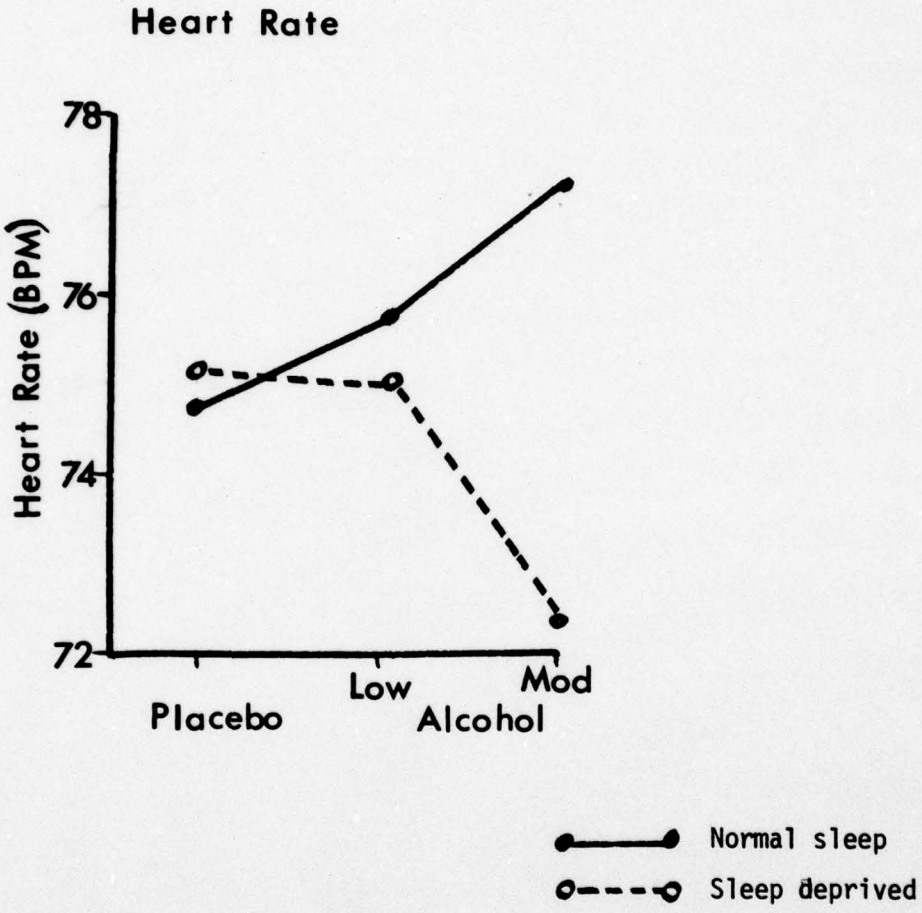


Figure 19. Study D. Mean alertness ratings for normal sleep vs sleep deprived conditions for each alcohol dose.

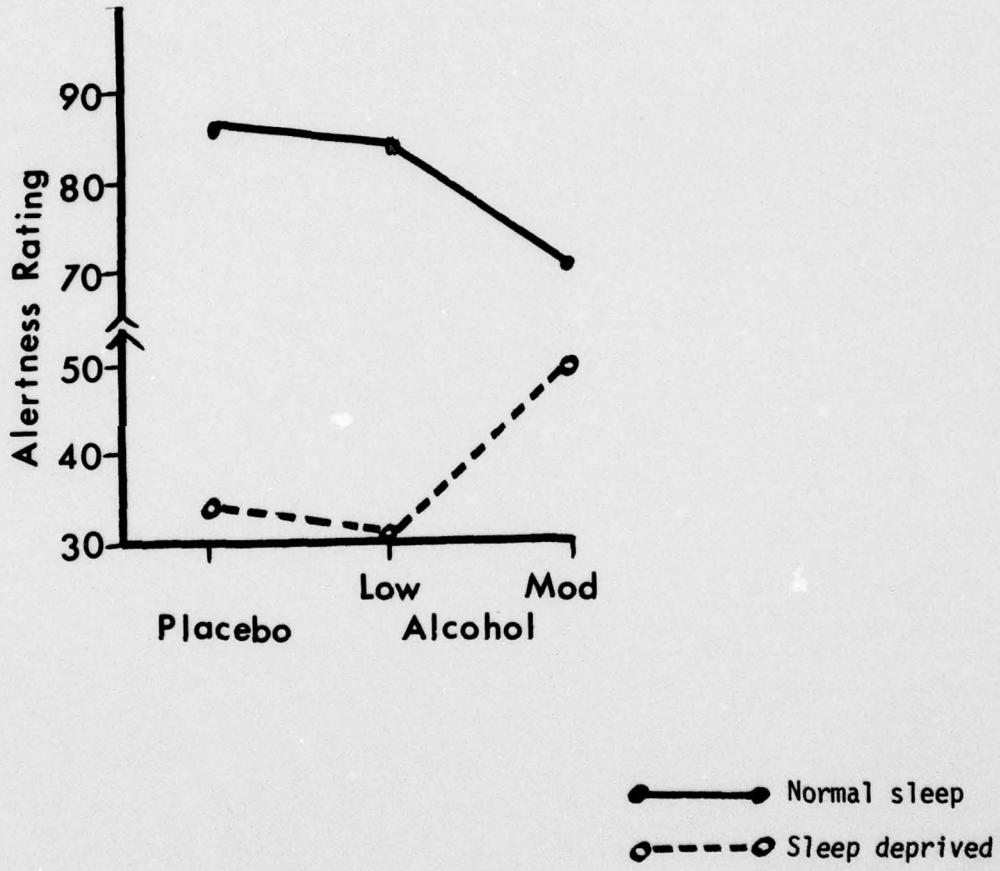
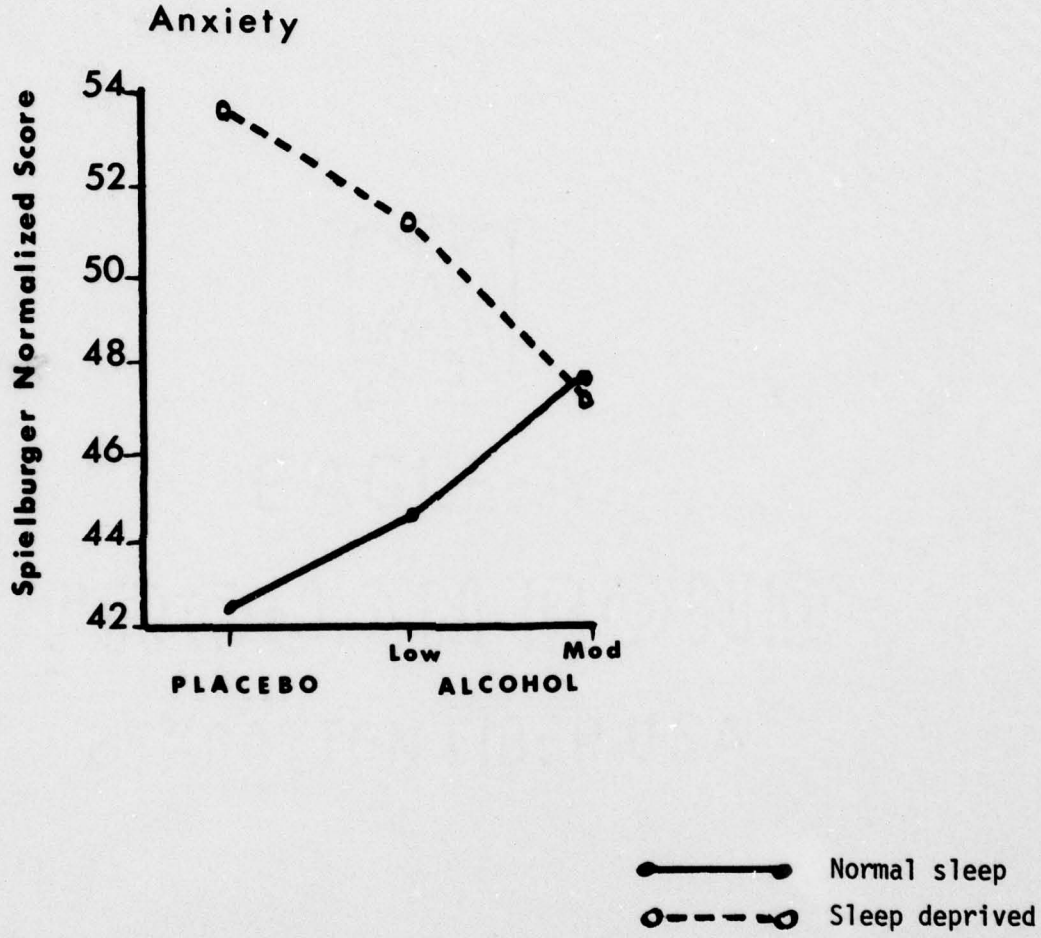


Figure 20. Study D. Mean anxiety ratings for normal sleep vs sleep deprived conditions for each alcohol dose.



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